Vertigo Academy International

II. Vertigo Academy International

22-23 May 2015

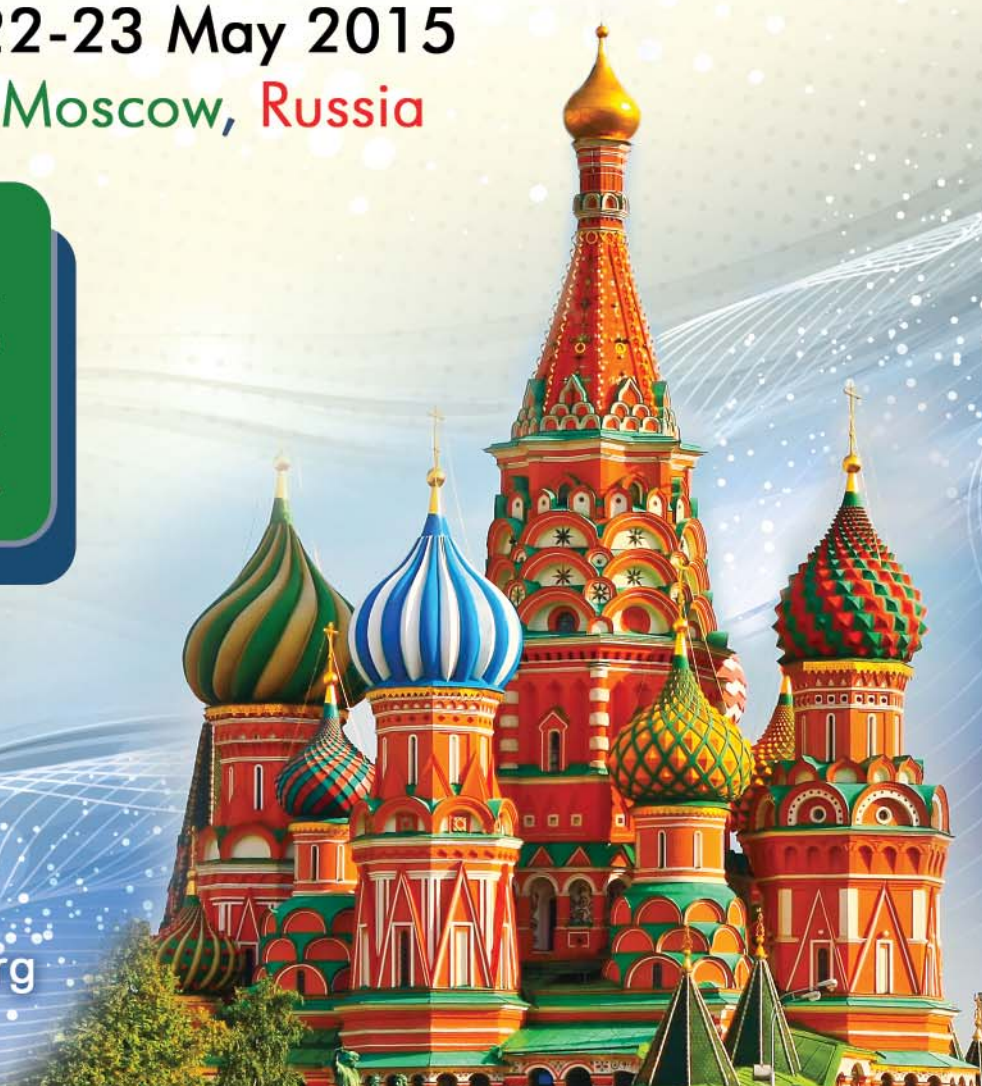
Moscow, Russia

PROGRAM & ABSTRACT BOOK

Under the Patronage of MSOA

MSOA

www.vainternational.org



EAONO

8th Instructional Workshop

European Academy of Otology & Neuro-Otology

Including

"Consensus in Auditory Implants"

28 September - 1 October 2016

Kaya İzmir Thermal & Convention

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References:

1) Serc® was registered in 1968 and first marketed (in Switzerland) in 1972
2) Legent F, Calais C, Cellier D. Vertiges paroxystiques itératifs et Serc. Étude clinique contrôlée. Concours Med 1988;110(29):2539–2543
3) Mira E, Guidetti G, Ghilardi L, et al. Betahistine dihydrochloride in the treatment of peripheral vestibular vertigo. Eur Arch Otorhinolaryngol 2003;260(2):73–77
4) Oosterveld WJ, Blijleven W, van Elferen LWM. Betahistine versus placebo in paroxysmal vertigo; a double blind trial. TGO Tijdschrift voor Therapie, Geneesmiddel, en Onderzoek 1989;14:122–126
5) Jeck-Thole S, Wagner W. Betahistine: a retrospective synopsis of safety data. Drug Saf 2006;29(11):1049–1059

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VO425 offers the same test protocols as VN415 and additionally expands into oculomotor tests with standard protocols for Gaze, Saccade, Smooth Pursuit and Optokinetic testing.

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EyeSeeCam Video Head Impulse Test (vHIT)

VNG Video Nystagmography

Nydiag Rotary Chair

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BOOK

II. Vertigo Academy International

22-23 May2015

Holiday Inn Sokolniki Hotel

MOSCOW - RUSSIA

www.vainternational.org

Contents

Dear Colleagues,

Finally we reached to the date we were working for, during the last 18 months. Even it was quite hard and intense, realizing this meeting with the contributions by the most prominent scientists of the world, gives me great honor and pleasure.

Vestibular topic constitutes only part of international otology meetings and it is quite rare to the topic to be emphasized on its own. I believe this meeting standing on with only vestibular topics will create a great impact. The scientific content as well as the contributors, of course is the main power for this impact.

Recently we have been the witness of new developments on the diagnosis of vestibular problems, but on the other hand, developing technologies to support the rehabilitation ideas for the bilaterally disabled patients as well as the changing habits on medical treatment regimes.

The "Vertigo Academy International" concept has began with the idea of putting the "Clinical Research and Basics" into the scientific agenda. In this regard the topics of the meeting has been chosen with great concern but also workshops are also added into the program to give practical issues to the participants.

I believe this will be a real up-to-date. The scientific content of the meeting will be documented and be shared further on

Now there appears a great interest and enthusiasm that stimulates us to continue on with the new "Vertigo Academy International" meetings, in another part of the world.

This meeting has been realized with the great support of ABBOTT Company. I know very well how ABBOTT Russia contributed to the organization with great self-sacrifice. But of course its Global Office was the main unit to give us great support and liberty.

In addition I wish to mention other companies (Interacoustics, Synapsys and Herinemann) who have also been part of the organizations.

The venue of the meeting is also so attractive with its fascinating historical background, and even if you have visited Moscow before, you will find it some other beautiful aspects still being in great enthusiasm.

Prof. Dr. O. Nuri Ozgirgin

Chairman

Vertigo Academy International

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Address: Rusakovskaya ul. Building 24 Moscow 107014, Russia
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The hotel is conveniently located 50 meters away from the metro station and 10 minutes walk from Sokolniki Exhibition Centre. Holiday Inn Moscow Sokolniki hotel offers 523 modern guest rooms of different categories - from comfortable standard to luxury suites including two executive floors and special rooms equipped for people with disabilities. Additional hotel facilities include free Wi-Fi (in all guest rooms, public areas and business center), and also fitness center, swimming pool, sauna and gym.

Sheremetyevo (SVO)

- Distance: 20.51 MI/33.0 km. North west to Hotel
- Time By Train : 30 minutes

Go along Leningradsky Shosse to ring road Mkad, turn east and go along Mkad. Turn to Schelkovskoye Shosse, drive along Schelkovskoye Shosse via Bolshaya Cherkizovskaya up to Rusakovskaya Str. The Hotel situated opposite Sokolniki Metro Station.

Vnukovo (VKO)

- Distance: 24.86 MI/40.0 km. South west to Hotel
- Time By Train : 40 minutes

Drive to Mkad turn to 3D ring and go right and go to the exit Ot Rusakovkaya Street. The Hotel is opposite Sokolniki Metro Station.

Domodedovo (DME)

- Distance: 27.96 MI/45.0 km. South to Hotel
- Time By Train : 40 minutes

Drive to Mkad turn to Kashirskoye Shosse, Drive along and turn right to Pr. Andropova. Drive along up to 3rd ring and turn right. Drive straignt to the exit to Rusakovskaya Street. The Hotel is on the right side opposite Sokolniki Metro.

Train

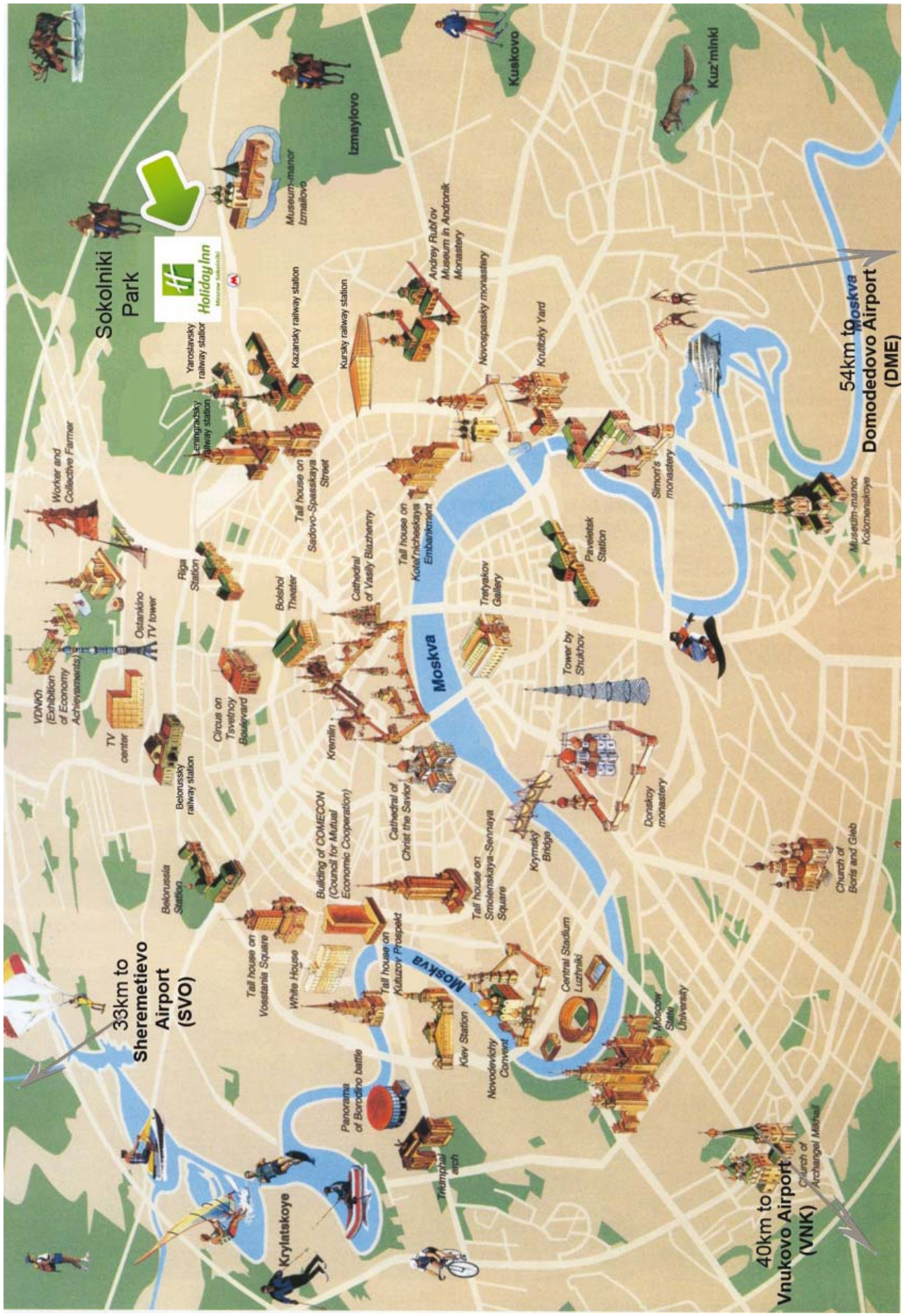
- Station Name : Leningradskiy Vokzal
- Distance: 1.06 MI/1.7 km. East to Hotel

From Komsomolskaya Square drive straight to Krasnoprudnaya street. Drive along Krasnoprudnaya street via Rusakovskaya street up to the hotel.

Subway

- Subway Station Name : Sokolniki
- Distance: 0.06 MI/0.1 km. North to Hotel

The hotel is located within a 5 minute walk, just across the street from Sokolniki subway station.


Sokolniki Park
Holiday Inn
Yaroslavsky railway station
Leningradsky railway station
Kazansky railway station
Kursky railway station
Museum-manor Izmailovo
Izmaylovo
Kuskovo
Kuz'minki
Andrey Rublyov Museum in Andronik Monastery
Novospassky monastery
Krutitzky Yard
Simon's monastery
54km to Moskva Domodedovo Airport (DME)
Museum-manor Kolomenskoye
Worker and Collective Farmer
Riga Station
Tall house on Sadovo-Spasskaya Street
Tall house on Kotel'nicheskaya Embankment
Paveletsk Station
Ostankino TV tower
VDNKh (Exhibition of Economy Achievements)
TV center
Belorussky railway station
Circus on Tsvetnoy Boulevard
Bolshoi Theater
Cathedral of Vasily Blazhenny
Kremlin
Tretyakov Gallery
Tower by Shukhov
Moskva
Belorussia Station
Building of COMECON (Council for Mutual Economic Cooperation)
Cathedral of Christ the Savior
Tall house on Smolenskaya-Sennaya Square
Krymsky Bridge
Donskoy monastery
Tall house on Vosstania Square
White House
Tall house on Kutuzov Prospekt
Central Stadium Luzhniki
Moscow State University
Church of Boris and Gleb
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MOSKVA
Kiev Station
Novodevichy Convent
Panorama of Borodino battle
Triumphal arch
Krylatskoye
Church of Archangel Mikhail
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Moscow Restaurant

The restaurant "Moskva" for up to 450 seats offers European and Russian buffet for breakfast, lunch and dinner. Take benefits of the culinary delicacies with a variety of salads, cold and hot appetizers and main dishes that suit any preferences.

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Orange Fitness was created as an answer to the ever growing demand in beauty and relaxing spaces.

Our specialists will assist you in finding the right fitness program according to your needs. In our fitness bar we serve drinks, light snacks and fresh fruits.

Swimming pool: Pool size: 10 x 20m. Water depth: 1.5 – 3.0m. Water temperature: 25°C

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Registration and Information Desk

Registration and information desks for the II.VAI International Moscow will be held at the Bar Passage area of the Holiday Inn Sokolniki Hotel during the following hours:

May 21, 2015	14:00 - 18:00
May 22, 2015	07:30 - 18:00
May 23, 2015	08:00 - 18:00

Official Language

Official language of the meeting and of correspondence is English. There will be two way (English-Russian / Russian-English) simultaneous translation in main session Sokolniki Hall.

Name Badges

Please wear your name badges at all times during the Meeting. Badges are color coded as follows:

Speaker, Organizing Committee Members	**: Red**
Participants	**: Dark Blue**
Serenas Tourism (Organizing Secretariat)	**: Orange**

Speakers' Room

Speakers should hand in presentations in the slide check and speakers' room as soon as possible after their arrival. The name is Okhotny Ryad and located ground floor in the Holiday Inn Sokolniki Hotel. Speakers must ensure that all files needed for the presentation are included in the media of their choice (CD, USB Device) and they should be tested on a computer other than that on which it was created. Prior to the scientific session, the authors should review their presentations to ensure that it transferred properly.

Certificate of Attendance

It will be given at the end of the congress from the Registration and Information Desk.

Internet Access

Internet cafes are available all around the city and wireless internet access for notebook and mobile device users at the Holiday Inn Sokolniki Hotel.

Mobile Phones

Delegates are kindly asked to verify that mobile phones are switched off (silent mode) during sessions, as a courtesy for speakers and attendees.

Coffee Breaks

Coffee breaks will be served in Sokolniki foyer at the coffee break times.

Lunch

Lunch will be served as open buffet at the Moscow Restaurantwith beverages on May 22nd – 23rd days.

Dinner

The dinner will be served open buffet inclusive of beverages at the Moscow restaurant on May 21st. Opening Ceremony Dinner with beverages will be served on 22 of May with entertainment program

Posters

The posters will be displayed between May 22-23, 2015 at the Bar Passage Area ofthe according to the following schedule:

Poster Mount Date & Time	: May 21, 2015 at 14:00
Poster Remove Date & Time	: May 23, 2015 at 17:00
Discussion Dates and Times	: May 22, 2015 at 11:00 - 11:30 & 16:00 - 16:30
	May 23, 2015 at 10:30 - 11:00 & 16:00 - 16:30

The organization secretariat is not responsible for the posters that haven't been removed after the session.

Poster discussions will take place in front of the posters at coffee break times.

Presenters should be beside their posters during the discussion dates and times.

Workshop Room

The workshop hall name is "Krimski val"and located at the ground floor of the Holiday Inn Sokolniki Hotel.

Holiday Inn Moscow Sokolniki

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	Theatre style	Classroom	Long Table	Banquet	Reception	m2	Dimensions	Height	Door H/W	Natural Daylight
Arbat	70	36	30	45	50	70.33	10,82 x 6,5	3.1	2 x 2,4	yes
Lubyanka	-	-	14	-	-	35.28	6,3 x 5,6	3.1	2 x 2,4	yes
Chistye Prudy	45	27	16	27	20	41.58	6,6 x 6,3	3.1	2 x 2,4	yes
Krasnye Vorota	65	36	22	45	40	59.85	9,5 x 6,3	3.1	2 x 2,4	yes
Ostozhenka	45	27	22	27	30	42.77	7,72 x 5,54	3.1	2 x 2,4	no
Krymsky Val	110	60	40*2	81	80	95.23	13,45 x 7,08	3.1	2 x 2,4	no
Okhotny Ryad	120	63	48	63	70	84.11	15,02 x 5,6	3.1	2 x 2,4	no
Sokolniki	660	285	88*3	369	750	504.22	28,2 x 17,88	7	2 x 2,15	no
Passage	-	-	-	115	1000	574.00	82 x 7	3.3	-	yes
Bar Passage	-	-	-	-	60	69.52	6,64 x 10,47	3.3	-	yes

Ground floor

ACTIVITY	LOCATION	FLOOR
Registration and Information Desk	Bar Passage	Ground Floor
Main Meeting Hall	Sokolniki Hall	Ground Floor
Workshop Room	Krimski val Hall	Ground Floor
Speakers' Room	Okhotniy ryad	Ground Floor
Exhibition Area	Sokolniki Foyer	Ground Floor
Poster Area	Passage	Ground Floor
Storage Room	Ostozhenka Hall	Ground Floor
Breakfast&Lunch&Dinner	Moscow Restaurant	2nd Floor

REGISTRATION AND INFORMATION DESK / Bar Passage

MAIN MEETING HALL / Sokolniki Hall

WORKSHOP ROOM / Krimski val Hall

SPEAKERS' ROOM / Okhotniy ryad

EXHIBITION AREA / Sokolniki Foyer

POSTER AREA / Passage

	MAIN HALL		WORKSHOP HALL
08:30-09:00	**Opening Ceremony**		
09:00-09:30	**SESSION – 1** **Chair:** Kemal Deger (Turkey) **LECTURE: UPDATE ON PHARMACOTHERAPHY PHARMACOTHERAPY OF VERTIGO, NYSTAGMUS AND CEREBELLAR ATAXIA: TRANSLATIONAL AND BACK-TRANSLATIONAL RESEARCH AND ONGOING CLINICAL TRIALS** **Speaker:** Michael Strupp (Germany)		
09:30-11:00	**PLENARY PANEL: PHARMOCOTHERAPHY OF VERTIGO AND NYSTAGMUS** **Moderator:** Armagan Incesulu (Turkey) **Panelists:** Sergei Lilenko (Russia), Alexander Amelin (Russia), Herminio Perez-Garrigues (Spain), Elena Sigaleva (Russia), Andreas Zwergal (Germany) ***SPEAKER PRESENTATIONS*** **Nystagmometry in assessment of the labyrinthine vestibular disturbances therapy efficacy** Sergei Lilenko (Russia) **Comparative efficacy of betahistine and cinnarizine treatment of vertigo in patients with migraine** Alexander Amelin (Russia) **Medical therapy in Meniere disease** Herminio Perez-Garrigues (Spain) **Efficacy of histaminergic drugs in experimental motion sickness** Elena Sigaleva (Russia) **Pharmacological modulation of central compensation after unilateral vestibulopathy: pros and cons** Andreas Zwergal (Germany)		
11:00-11:30	**Coffee Break**		
11:30-13:00	**SESSION – 2** **Chair:** Alexandra Guseva (Russia) **PANEL: BPPV – NEW INSIGHTS. HAVE WE REACHED TO CONSENSUS?** **Moderator:** Enis Alpin Güneri (Turkey) **Panelists:** Oleg Melnikov (Russia), Levent Özlüoğlu (Turkey), Ambrose Lee (Canada), Michel Troupet (France), Özlem Konukseven (Turkey) ***SPEAKER PRESENTATIONS*** **Benign paroxysmal positional vertigo comorbidity** Oleg Melnikov (Russia) **Horizontal semisircular canal BPPV** Levent Özlüoğlu (Turkey) **BPPV – New insights** Ambrose Lee (Canada) **Effect of repositioning maneuver type and postmaneuverrestrictions on vertigo and dizziness in benign positionalparoxysmal vertigo** Michel Troupet (France) **The efficiency of repositioning maneuvers, clues and pearls for a successful maneuvre for each variant of BPPV** Özlem Konukseven (Turkey)	11:30-13:00 11:30-12:00 12:00-12:30 12:30-13:00	**SESSION – 2** **Chair:** Catherine Blaivie (Belgium) **NEW VESTIBULAR AND VHIT DATA CASE STUDIES** **Speaker:** Michelle Petrak (USA) **DYNAMIC POSTUROGRAPHY: WHERE AND HOW DOES IT CONTRIBUTE?** **Speaker:** Michel Lacour (France) **HOW TO PERFORM AND INTERPRETE THE VHIT** **Speaker:** Laurent Tardivet(France)
13:00-14:00	**Lunch**		

MAIN HALL

14:00-15:30 **SESSION – 3**
Chair: Orhan Yılmaz (Turkey)

PLENARY SESSION: MANAGING THE CO-MORBIDITIES IN MENIERE'S DISEASE
Moderator: Tsutomu Nakashima (Japan)
Panelists: Badr el din Mostafa (Egypt), Marco Mandala(Italy), Ronen Perez (Israel), Eduard Matsnev (Russia), Sertac Yetiser (Turkey), Suchitra Prasansuk (Thailand)

15:30-16:00 **PLENARY LECTURE: Motion Sickness**
Speaker: John Golding (UK)

SPEAKER PRESENTATIONS

Meniere's disease and benign increased intracranial tension
Badr el din Mostafa (Egypt)

Meniere's disease and vestibular migraine
Marco Mandala(Italy)

Bilateral Meniere's disease
Ronen Perez (Israel)

Otolith disfunctions at early and late stages in Meniere's disease
Eduard Matsnev (Russia)

Benign paroxysmal positional vertigo associated with Meniere's disease
Sertaç Yetişer (Turkey)

Prevalence of systemic autoimmune disorders as co-morbidity in MD
Suchitra Prasansuk (Thailand)

16:00-16:30 **Coffee Break**

16:30-17:00 **SESSION – 4**
Chair: Ismet Bayramoglu (Turkey)

LECTURE: SUPERIOR CANAL DEHISCENCE
Speaker: Issam Saliba (Canada)

17:00-18:30 **PANEL: TREATMENT OF MENIERE'S DISEASE RESISTANT TO CONSERVATIVE TREATMENT**
Moderator: Mans Magnusson (Sweden)
Panelists: Tayfun Kirazlı (Turkey), Franco Trabalzini (Italy), Sofiane Ouhab (Algeria), Maurizio Barbara (Italy), Prepageran Narayanan (Malaysia)

SPEAKER PRESENTATIONS

Single-shot, low-dose intratympanic gentamicin in Ménière disease: role of vestibular-evoked myogenic potentials and caloric test in the prediction of outcome
Tayfun Kirazlı

Evaluation of surgical treatment for disabling vertigo in unilateral Meniere's disease
Franco Trabalzini (Italy)

Surgical option when ménière's disease other treatments fail
Sofiane Ouhab (Algeria)

Pressure treatment for recalcitrant ménière's disease
Maurizio Barbara (Italy)

Surgery for vertigo
Prepageran Narayanan (Malaysia)

WORKSHOP HALL

16:30-17:00 **SESSION – 4**
Chair: Sergey Kosyakov (Russia)

Intratympanic applications
Speakers: Sergey Kosyakov (Russia), Saba Battelino (Slovenia)

SPEAKER PRESENTATIONS

Intratympanic steroids for Meniere disease, preliminary results
Sergey Kosyakov (Russia)

Intratympanic (transtympanic) multiple corticosteroid applications in patients with non surgical therapy resistant Meniere disease
Saba Battelino (Slovenia)

17:30-17:30 **Acute vestibular loss: management**
Speakers: F. Necdet Ardıç (Turkey), Cem Bilgen (Turkey)

SPEAKER PRESENTATIONS

Acute vestibular loss: Management part I
F. Necdet Ardıç (Turkey)

Acute vestibular loss and the treatment
Cem Bilgen (Turkey)

17:30-18:00 **How to diagnose and treat bppv of any canal**
Speaker: Daniel Nuti (Italy)

18:00-18:30 **Nystagmus of central origin**
Speakers: Ossama Mansour (Egypt), Amr Gouda (Egypt)

	MAIN HALL		WORKSHOP HALL
08:30-09:00	**SESSION – 5** **Chair:** Alla Guekht (Russia) **PLENARY LECTURE: FROM THE EARS TO THE EYES FROM THE EARS TO THE EYES: USING THE BRAIN IN BETWEEN TO SHARPEN OUR DIAGNOSTIC TOOLS** **Speaker:** David Zee (USA)		
09:00-10:30	**PLENARY SESSION: NON-MENIERE PERIPHERAL VESTIBULAR DISEASE** **Moderator:** O. Nuri Ozgirgin (Turkey) **Panelists:** Nazım Korkut (Turkey), Ossama Hamid (Egypt), Antonio Lopez Escamez (Spain), Natalia Kunelskaya (Russia), Avi Shupak (Israel) ***SPEAKER PRESENTATIONS*** **Perilymph fistula** Nazım Korkut (Turkey) **Genetics of peripheral vestibular disorders** Antonio Lopez Escamez (Spain) **Ménière's-like vestibular disorders** Natalia Kunelskaya (Russia) **Prednisone treatment for vestibular neuritis** Avi Shupak (Israel)		
10:30-11:00	**Coffee Break**		
11:00-11:30	**SESSION – 6** **Chair:** Onur Odabaşı (Turkey) **CENTRAL AND PERIPHERAL VESTIBULAR DISORDERS** **Speakers:** Mete Kiroglu (Turkey), Vladimir Parfenov (Russia)	11:00-11:30	**SESSION – 6** **Chair:** Mario Milkov (Bulgaria)
11:30-13:00	***SPEAKER PRESENTATIONS*** **Central and peripheral vestibular disorders** Mete Kiroglu (Turkey) **The diagnosis of central and peripheral vestibular disorders in outpatients department** Vladimir Parfenov (Russia) **PANEL: VESTIBULAR MIGRAINE** **Moderator:** Mark Obermann (Germany) **Panelists:** Anirban Biswas (India),Nese Çelebisoy (Turkey), Onur Çelik (Turkey), David Zee (USA), Timothy Hain (USA), ***SPEAKER PRESENTATIONS*** **Migraine related vertigo covering differential diagnosis, treatment options and relationship with migraine** Anirban Biswas (India) **Medical treatment of vestibular migraine** Nese Çelebisoy (Turkey) **Key points of patient's history and roles of vestibular tests for vestibular migraine diagnosis** Onur Çelik (Turkey) **Vestibular migraine panel** David Zee (USA) **Migraine and dizziness** Timothy Hain (USA)	11:30-12:00	**A NEW ERA IN THE TREATMENT OF MENIERE'S DISEASE: THE ENDOLYMPHATIC DUCT BLOCKAGE.** **Speakers:** Issam Saliba 11:30-12:00 **VNG INTERPRETATION** **Speaker:** Bernard Cohen (France) 12:00-12:30 **Meniere disease – New horizons in diagnosis** **Speaker:** Paul Avan (France) 12:30-13:00 **Cerebellar infarctions mimick acute peripheral vertigo** **Speaker:** Maxim Zamergrad (Russia)
13:00-14:00	**Lunch**		

MAIN HALL			WORKSHOP HALL
14:00-14:30	**SESSION – 7** **Chair:** Sylviu Albu (Romania) **LECTURE: NEUROPHYSIOLOGY OF VESTIBULAR REHABILITATION** **Speaker:** Timothy Hain (USA)	14:00-14:30	**SESSION – 7** **Chair:** Tuncay Özçelik (Turkey) **Bedside examination** **Speakers:** Ali Ozdek (Turkey), Bulent Satar (Turkey)
14:30-16:00	**PANEL: VESTIBULAR COMPENSATION AND REHABILITATION** **Moderator:** Jacques Magnan (France) **Panelists:** Herman Kingma (Netherlands), Michel Lacour (France), François Caces (France), Carolina Binetti (Argentina) ***SPEAKER PRESENTATIONS*** **Vestibular compensation and rehabilitation** Herman Kingma (Netherlands) **Panel on vestibular compensation and rehabilitation** Michel Lacour (France) **Vestibular compensation after vestibular neurotomy** François Caces (France) **Vor rehabilitation with head thrust for vestibular compensation** Carolina Binetti (Argentina)	14:30-15:00 15:00-15:30 15:30-16:00	**Vertical nystagmus – what does it mean?** **Speaker:** Ji-Soo Kim (South Korea) **Vestibular interaction of cochlear implantation** **Speaker:** Antonio della Volpe (Italy) **Vestibular problems in pediatric population** **Speaker:** Wiener Vacher (France)
16:30-17:00	**SESSION – 8** **Chair:** O. Nuri Ozgirgin (Turkey) **Plenary lecture: expectations for the future** **Speaker:** Charles della Santina (USA)		
17:00-18:00	**Plenary session: messages to take home** **Moderator:** Ji-Soo Kim (South Korea) **Panelist:** Maurizio Barbara (Italy), Mans Magnusson (Sweden), Jacques Magnan (France), Mohan Kameswaran (India)		

FACULTY ABSTRACTS

22 MAY 2015 / MAIN HALL

09:00-09:30

PHARMACOTHERAPY OF VERTIGO, NYSTAGMUS AND CEREBELLAR ATAXIA: TRANSLATIONAL AND BACK-TRANSLATIONAL RESEARCH AND ONGOING CLINICAL TRIALS

Michael Strupp, Andreas Zwergal
University of Munich, Department of Neurology and German Center for Vertigo and Balance disorders, 81377 Munich, Germany

For the pharmacotherapy of vertigo and dizziness there are basically eight groups of drugs that can be used (the "8 'A's"): anti-emetics; anti-inflammatory, anti-Menières, and anti-migraineous medications; anti-depressants, anti-convulsants, aminopyridines and acetyl-DL-leucine.

Symptomatic treatment of acute vertigo

Recently it was shown in an animal model of acute unilateral vestibulopathy that the potassium channel blocker 4-aminopyridine reduced postural imbalance already 30 min after ingestion of the drug; it might therefore be ideal for the symptomatic treatment of acute attacks of vertigo. Acetyl-DL-leucine, a modified amino acid, improved central compensation in this animal model. Using the same model, it was demonstrated by micro-PET that betahistine increased glucose metabolism of the vestibular nuclei ipsilateral to the lesion on days 1 and 15 after labyrinthectomy; thereby it can reduce the symptoms and improve central compensation without a sedative effect. These effects of betahistine are currently being evaluated in a randomized-controlled trial (RCT, the BETAVEST-trial).

Peripheral vestibular disorders

Acute unilateral vestibulopathy. An RCT showed that methylprednisolone alone significantly improves the recovery of peripheral vestibular function in thus affected patients. There is, however, not sufficient current evidence for a general recommendation.

Menière's disease (MD). Currently we do not have sufficient evidence that 24 mg twice per day or 48 mg tid betahistine-dihydrochloride have a significant effect on the number of Menière's attacks. Based on clinical experience, higher dosages of up to 1440 mg per day are now being evaluated to "titrate" the attacks; in animal studies it was shown that betahistine increases inner ear blood flow in a dose-dependent manner by its action as an inverse-agonist of the H3 receptor.

Vestibular paroxysmia. Vestibular paroxysmia is characterized by recurrent brief attacks of vertigo due to a neuro-vascular cross-compression of the vestibular nerve. In a randomized controlled trial it was shown that oxcarbazepine reduces the number of attacks of vestibular paroxysmia. There is an on-going RCT with carbamazepine (the VesPa-trial).

Central nystagmus, cerebellar disorders and vestibular migraine

Downbeat and upbeat nystagmus and episodic ataxia type 2. Recent trials showed that 4-aminopyridine (in well tolerated dosages of 5 to 10 mg tid) is effective for the treatment of downbeat nystagmus and upbeat nystagmus. It was also demonstrated in an RCT that 4-aminopyridine significantly reduces the attacks of ataxia in patients with episodic ataxia type 2 (EA 2). The effects of 4-aminopyridine and acetazolamide are currently being studied in the EAT-2-TREAT-trial in EA 2.

Cerebellar ataxia.Acetyl-DL-leucine improves cerebellar ataxia (three observational studies); these finding are the basis for an on-going multinational RCT (the ALCAT-trial). 4-aminopyridine reduces gait variability in thus affected patients which is also evaluated in an RCT (the FACET-trial).

Vestibular migraine. Currently, no specific therapy either for the attacks or for the prophylactic treatment can be recommended. The effects of metoprolol are being evaluated in an RCT (the PROVEMIG-trial)

09:30-11:00

NYSTAGMOMETRY IN ASSESSMENT OF THE LABYRINTHINE VESTIBULAR DISTURBANCES THERAPY EFFICACY

Sergei Lilenko
Head of Vestibular Disorders Laboratory of the St Petersburg Ear, Nose, Throat and Speech Research InstituteProfessor of Otolaryngology Departmentin the North-West Medical University
Doctor of Medical Science, St Petersburg, Russian Federation

On ample factual material (274 patients), we studied ear labyrinth receptors pathology that is determined by different factors (vascular dysfunction, degeneration, inflammatory alterations, metabolic disturbances, trauma and mechanical effect). To assess the extent of vestibular dysfunction we used authentic computer electrooculography technique. Peripheral type of spontaneous nystagmus, labyrinth asymmetry in caloric trials and total visual suppression of caloric nystagmus confirm the diagnosis of vestibular system alteration on the level of vestibular receptors. Patients were given nosotropic therapy for the revealed acute vascular labyrinthopathy, ototoxicity, and different forms of labyrinthitis, Meniere's disease, posttraumatic labyrinthopathy and perilymphatic fistula. Nystagmometric technique with automatic evaluation of nystagmus dynamic characteristics (amplitude, frequency, slow component velocity) makes the assessment of treatment efficacy possible. Vanishing of spontaneous and pressure nystagmus, absence of optokinetic nystagmus asymmetry, decrease of labyrinth asymmetry in caloric tests after complex medicated therapy or after unilateral ear surgery objectifies the disappearance or relief of vestibular disturbances. In cases of persisting complaints on vertigo or signs of vestibular function failure, revealed by means of computer electrooculography, vertigolytic therapy enhancement is required. In acerbation of the labyrinthine vestibular disturbances, patients were assigned to active therapy (rehabilitation exercises, Betahistine dihydrochloride taking in complex with glucocorticoid, diuretic and tranquilizer). Diminishment or completely stopping the gyration sensation and ceasing of nausea / vomiting and other vegetative symptoms of acute attacks should be achieved as quickly as possible by using any neurotropic or antihistamine drug or anticholinergic agent with vestibular suppressant and antiemetic effects.

COMPARATIVE EFFICACY OF BETAHISTINE AND CINNARIZINE TREATMENT OF VERTIGO IN PATIENTS WITH MIGRAINE

Alexander Amelin , L.E. Babayan, Anna Kudryavtseva

Headache and vertigo are the most common complaints, and they often emerge simultaneously. Fifty-six (40%) patients complaining of vertigo out of 140 patients with migraine were studied. Emerging in the aura and/or in headache phases, vertigo was more frequently registered in patients suffered from migraine with aura (57%). Vertigo associated with migraine was diagnosed in 25% of the cases. The patients were randomized into 2 equal identical groups, one of which was treated by betahistine (16 mg, 3 times daily before meal) and the other one was given cinnarizine (25 mg, 3 times daily). Treatment duration was 12 weeks. Reduction of vertigo attacks frequency and headache by 50% and over, in comparison to the baseline period, was considered as beneficial. Fifty-three (95%) patients completed the treatment course. Decrease of a risk of negative results and a frequency of positive effect of vertigo therapy were significantly higher in the group receiving betahistine (p=0.492). Reduction of monthly relapses by 50% and over was detected in 79% of the patients of betahistine group and in 52% of those of cinnarizine one (p=0.492, Fisher's exact test). Migraine attacks monthly frequency was diminished by 43% and 64%, respectively. However, these differences were not statistically significant (p=0.170). Therefore, betahistine is considered for using as vertigolytic medication and for migraine attacks prevention.

Key words: migraine, vertigo, betahistine, cinnarizine.

MEDICAL THERAPY IN MENIERE DISEASE

Herminio Pérez Garrigues
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Typical symptoms in Meniere Disease are attacks of dizziness, hearing loss, and a feeling of fullness in the ear. Nevertheless there are some other symptoms that can be relevant in certain circumstances such as instability. Also, the uncertainty of when a new dizziness attack will come may limit the patient's life. It is not rare that Meniere patients develop depression or neurotic vestibular disorder. Medical treatment must look at all these problems.

We have been using some software to follow up and control our Meniere patients. More than 400 cases have been registered and are being followed up in our data base. Through this software we can easily visualize the evolution of symptoms and treatment response.

Our treatment strategy is based on appeasing the illness. In order to achieve that, a complete and clear explanation of what Meniere Illness is, is given to each patient. We also insist on the fact that medical help will be provided any time it is needed.

Each patient is given a form at the first visit on which they keep a record of every dizziness attack describing length, accompanying vegetative symptoms, migraine, stress situation, tinnitus evolution, etc.

Regularly the first target is to prevent dizziness attacks. Less intervention possibilities are available in the audition and tinnitus fields. A satisfactory result would be that the patient is able to get along with his normal life with no fear of dizziness attacks.

If only a few dizziness crisis occur, a specific dietary regimen associated with vestibular suppressant drugs during the attack can be provided. In case of frequent dizziness attacks a basal treatment with Betahistine, diuretics or both is arranged. Betahistine dihydrochloride is a histamine-like drug widely used in relieving the symptoms associated with Ménière's syndrome. In bilateral cases oral steroid therapy is considered.

If no response is observed, intratympanic steroids can be an alternative, nevertheless it is not guaranteed and some patients can keep experiencing dizziness attacks, so intratympanic Genatamicine can be used.

Anxyolitic and antidepressant treatment may benefit some patients.

In our presentation we will discuss treatment instructions and outcome, in addition to strategies to compare results.

EFFICACY OF HISTAMINERGIC DRUGS IN EXPERIMENTAL MOTION SICKNESS

Elena Sigaleva, Eduard Matsnev
Department of Physiology and Pathology of Auditory and Vestibular Systems, Federal Scientific Center (FSC), Institute for Biomedical Problems, Russian Academy of Sciences (RAS), Moscow, Russia

The purpose of our preclinical pilot study was to evaluate the efficacy of the histaminergic drug betahistine dihydrochloride for prophylaxis of motion sickness symptoms. 10 healthy volunteers (mean age 19.4 y.o.) with high susceptibility to motion sickness participated in the study modeling experimental motion sickness. Motion sickness was modeled using Coriolis (precession) accelerations (cumulative Coriolis stimulation test – CCST). Each subject took 32 mg of "Betahistine dihydrochloride" or placebo under "double – blind" conditions 1 hour before testing. The duration and slow phase velocity of the post-rotational nystagmus, the pursuit eye tracking test, and the latency, velocity and accuracy of saccades were estimated. The tolerability level of the CCST in volunteers in the betahistine series was shown to be significantly ($p < 0.001$) higher, as compared to placebo and baseline. The mean illusory sensations score for the experimental series was significantly lower than that in the placebo and baseline series ($p < 0.01$). It was found that "Betahistine" demonstrated antimotion sickness efficacy and improved oculomotor activity (increased gain during pursuit movements, faster and more accurate saccades).

Betahistine dihydrochloride is known to act as an H1receptor agonist and as a partial H2 receptor agonist. It also interacts with the histamine receptors that modify histaminergic neurotransmission in the brain. Studies carried out in recent years have allowed identification of direct projections of the tuberomamillary histaminergic neurons on the vestibular nuclear complex. Takeda et al. show from their studies the presence of histaminergic projections from the posterior hypothalamus to the medial vestibular nuclei in rats, and underline that these connections may have implications for motion sickness.

The so-called "sensory conflict" (mismatch) theory of motion sickness development is based on the notion that the triggering stimuli of the motion in motion sickness, after they are perceived by the semicircular canals, otolith organs, vision, and somatosensory receptors, proceed to the brainstem structures and are

"recognized" by a specific nervous centre, the "comparator". After that, this information is compared to the "sensory image" held by the memory on the basis of previous "sensory experience" acquired by the body in the process of its development. In case the "comparator" produces output sensory information that is in conflict with that held by the brain's memory, nervous processes are activated that initiate the development of symptoms and signs of motion sickness. Traditionally, three classes of anti-motion sickness medications are used: drugs that block the sensory input responsible for the "sensory conflict" (mismatch); agents that modify the "nervous model" to new signals from the sensory input; drugs that inhibit the mechanisms in control of the symptoms and signs of motion sickness. The possible mechanisms of the protective effect of betahistine in motion sickness are discussed.

The reported experiments thus demonstrated the promise of the possible use of betahistine dihydrochloride as a preventive means for motion sickness, as well as the relevance of further studies in this area.

PHARMACOLOGICAL MODULATION OF CENTRAL COMPENSATION AFTER UNILATERAL VESTIBULOPATHY: PROS AND CONS

Andreas Zwergal
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An acute unilateral vestibular lesion leads to a vestibular tone imbalance with nystagmus, head roll tilt and postural imbalance. These deficits gradually decrease over days to weeks due to central vestibular compensation (VC). Pharmacological therapy in acute unilateral vestibular disorders may be either guided by the aim to reduce symptoms or to improve the course of VC.

Pharmacological treatment of symptoms in acute unilateral vestibulopathy

In clinical practice several drugs (mainly antiemetics, hypnotics) are used to reduce vegetative symptoms of vestibular imbalance. These agents interact with histamine, dopamine, serotonine or GABA receptors in the brainstem and cerebellum. However, early symptomatic treatment of acute unilateral vestibulopathy is thought to impede the course of ensuing VC. Despite the great clinical importance of this hypothesis there is no experimental evidence of its validity. Recently it was shown in an rat model of acute unilateral vestibulopathy that 1) the administration of the potassium channel blocker 4-aminopyridine 4-AP transiently improves postural imbalance in acute unilateral vestibulopathy by activating the vestibulocerebellum; 2) 4-AP-induced reduction of the symptom burden during the acute phase of the vestibular syndrome impedes the subsequent course of postural compensation; the individual symptomatic response to 4-AP strongly predicts the postural outcome during compensation; 3) The cerebral correlate of this impaired compensation is a prolonged asymmetry between the vestibular nuclei and posterolateral thalami after 4-AP administration. These results support the hypothesis that the symptomatic pressure in the initial phase of an acute vestibular syndrome essentially drives the mechanisms of VC. Therefore, symptomatic treatment should only be given on demand and for a short time. It is likely that this statement also applies for other symptomic treatments like antiemetics or sedative drugs.

Pharmacological improvement of vestibular compensation after unilateral vestibulopathy

An alternative therapeutic strategy is the application of drugs, which shall improve and accelerate VC after an acute unilateral vestibulopathy. In the past several pharmacological agents (e.g. N-acetyl-DL-leucine, betahistine, corticosteroids) have been proposed. Their mode of action however is not completely understood and there are only few controlled prospective clinical trails to prove their efficacy in patients. In a rat model of unilateral labyrinthectomy we could recently show that 1) N-acetyl-DL-leucine accelerates the postural compensation after unilateral vestibular damage; 2) N-acetyl-L-leucine is the pharmacologically active enantiomer that induces this effect; 3) the potential mechanism of N-acetyl-L-leucine action for improving vestibular compensation consists of an activation of the vestibulocerebellum and a deactivation of the posterolateral thalamus; 4) N-acetyl-L-leucine effects thalamic and cerebellar metabolism only after peripheral vestibular but not auditory damage. Few studies animal studies suggest a beneficial effect of betahistine on VC. The German Center for Vertigo and Balance Disorders currenly runs a multicenter prospective controlled clinical trial on the effect of betahistine on VC after acute vestibulopathy (BETAVEST-trial). A positive effect of corticosteroids on VC has been assumed, but a strict prove is lacking. In the setting of vestibular neuritis several clinical studies have shown a benefit of corticosteroids for the peripheral vestibular outcome and functional compensation.

11:30-13:00

BENIGN PAROXYSMAL POSITIONAL VERTIGO COMORBIDITY

O.A. Melnikov
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Up to the present moment causes of BPPV development remain unknown despite the fact that this disease is the most common cause of the vestibular vertigo.

Interest to study BPPV comorbidity is due to possibility to find the most common combinations with other diseases which can provide an answer to questions about reasons for BPPV, find risk groups of development of this illness or determine patterns for observance and treatment of patients with different diseases.

Idiopathic BPPV which does not allow to determine the disease cause develops in 75% of cases. In other cases there is some cause to which BPPV onset can be related. The latter is regarded as secondary disease in comparison with the idiopathic form. Reasons of the secondary BPPV in 25% of cases can be head traumas, labyrinthitis, Meniere's disease, surgical operations in middle ear, respiratory viral infections, etc.

Heredity is possible to develop of BPPV. Although specific genes responsible for this disease have so far not been found, it has been established that patients with BPPV were 5 times as likely to have relative with BPPV compared to the control group (Gizzi M. 1998).

According to some authors, subjects with BPPV have high rates of diabetes, ear/hearing problems, thyroid problems,

allergies, high cholesterol, autoimmune thyroiditis headaches, arteritis, hyperuricemia, hormone imbalance, mild head trauma and sinus diseases. Balance was impaired worse in diabetics (Cohen H.S. et all 2004, Ogun O.A. 2014, Modugno G.C. et all 2000, Amor-Dorado J.C. et all 2004).

BPPV can be caused by head or ears traumas, though this group is not significant in comparison with incidence of idiopathic BPPV. In the post-traumatic group there were no gender differences in patients, bilateral involvement was more prevalent (Katsarkas A. et all 1999).

BPPV is observed in patients with different ear pathology. BPPV was found in association with idiopathic sudden deafness in 50.7%, with Meniere's disease in 28.9%, with vestibular neuritis and herpes zoster oticus in 20.2% (Lee N.H. 2010). BPPV with vestibular neuritis appears to take more time to treat than idiopathic BPPV (Nuti D. et all 2012).

It was found that BPPV incidence increases if compared with the similar population of patients with migraine. At the same time, the clinical pattern of BPPV was absolutely typical and repositioning maneuvers were effective. Migraine was 3 times more common in patients with idiopathic BPPV than in patients with a certain cause of BPPV – either a trauma or surgical intervention in ear. Presumably, BPPV is recurrent in patients with migraine due to some pathology in the inner ear, probably affecting vascular supply (Ishiyama A. et all 2000).

Presently a number of studies evidence some correlation between BPPV development and osteoporosis, and some studies provide systemic analysis of the information gathered (Yu S. et all, 2014). The incidence of BPPV recurrence increases in patients with osteoporosis. According to Yamanaka T. et all 2013, the incidence of BPPV recurrence in this group was 56.3%, which was significantly higher than in patients with normal bone mineral density (16.1%). Furthermore, the frequency of BPPV recurrence increases as bone mineral density decreased.

Vitamin D plays an important role in osteoporosis and osteopenia as it influences regulation of both calcium and phosphorus, which finally maintains proper bone structure. Decreased of vitamin D level were registered in BPPV patients in comparison with the control group (Jeong S.H. et all 2013).

Thus, secondary BPPV can have different associations with other diseases which can have the same etiology or significantly affect the BPPV progress. In our report we would attempt to share our opinion on this matter.

HORIZONTAL SEMISIRCULAR CANAL BPPV:

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Department of ORL-HNSBaskent University School of Medicine, Ankara, Turkey

Abstract: Since the first description of Horizontal Semiscircular Canal BPPV (H-BPPV) by McClure (1985) and detailed description by Pagnini (1989) and Baloh (1993) it is stated that H-BPPV accounts for 5-20 % of all BPPV cases. Main complaint is vertigo while lying and Rolling in the bed. Patients describe brief vertigo episodes and vertigo is more often seen on the affected side.

Etiology: BPPV may be seen as a isolated complaint, while also accompany to the patients with head trauma, labyrinthitis, vestibular neuritis, Meniere's disease and migrain.

Diagnosis: History of the patient is important. There are Diagnostic test for H-BPPV. Supine Roll Test is made while the patient in supine position and head slightly elevated to put horizontal SC to vertical position with respect to ground. The head is then quickly turned 90 degrees to one side, then to center position and turned to opposite side in order.

There are two types of typical nystagmus to diagnose a H-BPPV. Geotropic variant, where eyes beat towards to lower ear, is seen in patients with canalolithiasis. A slower form of Geotropic nystagmus will also be recorded when the head is turned towards to uneffected ear. Second form of H-BPPV Nystagmus is called Ageotropic variant. It is recorded on both head positions but nystagmus is stronger when head is turned to the unaffected side.

The differential diagnosis of Canalolithiasis and cupulolithiasis of H-BPPV is based on the Ewald's second Law (1882). This is "In the horizontal semicircular canals, an ampullopetal endolymph movement cases a greater stimulation than an ampullofugal one." In the case of canalolithiasis: When the head is turned to the affected side canaliths move towards to ampulla and this creats an excitation, where moving to the opposite side causes clot move away from the cupula and causes an inhibition and weaker nystagmus. In the case of cupulolithiasis: When the head is turned to unaffected side heavy cupula moves towards to ground and away fri-om the HSC and causes stronger nystagmus, and when the head is moved to the affected side heavy cupula moves towards to HSC and this causes an inhibition which causes weaker nystagmus compared to the unaffected side.

Treatment: There are several particle repositioning maneuvers (PRP) reccomended by different authors (Vanucchi, Epley, Lempert and Tiel-Wick. In addition to PRPs there are also habituation exercises.

A COUPLE OF PATIENTS WITH H-BPPV WILL ALSO BE PRESENTED DURING THE PRESENTATION.

BPPV – NEW INSIGHTS

Ambrose Lee
Canada

Here is a summary what I intend to bring to the discussion table.

A review of the current pathophysiology (canalolithiasis, cupulolithiasis, and canalith jam), diagnosis and treatment of posterior canal, anterior canal and horizontal canal BPPV.

The usage of other ancillary tests such as oVEMP, MRI scans in their diagnosis.

Relationship between Superior canal dehiscence, vestibular neuronitis, Meniere's disease and BPPV.

Post BPPV syndrome

Surgery for BPPV

EFFECT OF REPOSITIONING MANEUVER TYPE AND POSTMANEUVERRESTRICTIONS ON VERTIGO AND DIZZINESS IN BENIGN POSITIONALPAROXYSMAL VERTIGO

Michel Toupet[1], Alexis Bozorg Grayeli[2]
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Introduction: To compare the efficiency of Epley (Ep) and Sémont-Toupet (ST) repositioning maneuvers and to evaluate postmaneuver restriction effect on short-term vertigo and dizziness after repositioning maneuvers by an analog visual scale (VAS) in benign positional paroxysmal vertigo (BPPV).

Material and Methods: 226 consecutive adult patients with posterior canal BPPVwere included. Patients were randomized into2 different maneuver sequence groups (n = 113): 2 ST then 1 Ep or 2 Ep then 1 ST.

Each group of sequence was randomized into 2 subgroups: with or without postmaneuver restrictions. Vertigo and dizziness were assessed from days 0 to 5 by VAS. Results. There was no difference between vertigo scores between Ep and ST groups. Dizzinessscores were higher in Ep group during the first 3 days but became similar to those of ST group at days 4 and 5. ST maneuversinduced liberatory signs more frequently than Ep (58% versus 42% resp., P <0.01, Fisher's test). After repositioning maneuvers,VAS scores decreased similarly in patients with and without liberatory signs. Postmaneuver restrictions did not influence VASscores.

Conclusion: Even if ST showed a higher rate of liberatory signs than Ep in this series, VAS scores were not influenced bythese signs.

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Toupet M, Ferrary E, Bozorg Grayeli A. Effect Of Repositioning Maneuver Type And Postmaneuver Restrictions On Vertigo And Dizziness In Benign Positional Paroxysmal Vertigo. ScientificWorldJournal. 2012;2012:162123.

THE EFFICIENCY OF REPOSITIONING MANEUVERS, CLUES AND PEARLS FOR A SUCCESSFUL MANEUVRE FOR EACH VARIANT OF BPPV

Ozlem Konukseven

Director of Hearing & Balance Disorders, Diagnose and Rehabilitation Center, Karatay University, Konya, Turkey Associate Professor in Karatay University Faculty of Health Science, Division of Audiology, Konya, Turkey

This presentation is designed to answer questions " What the ergonomic treatment is for BPPV " and "clues and pearls for a succesful maneuvre can be explained with anotomy and physiology of SCC

14:00-15:30

MENIERE'S DISEASE AND BENIGN INCREASED INTRACRANIAL TENSION

Badr el Mostafa
Ain-Shams University, Cairo, Egypt

Rationale: Meniere's disease is thought to be pathophysiologically due to increased pressure in the endolymphatic spaces leading to distortion of the sensory elements. As the inner ear fluids are in direct and indirect contact with CSF, it was hypothesized that changes in CSF hydrodynamics could affect inner ear fluid pressures.

Patients and methods: This study was conducted on 50 patients presenting with benign increased intracranial tension diagnosed by Dandy's criteria and by radiological data. All patients were subjected to a detailed vertigo questionnaire and underwent comprehensive clinical, audiological and vestibular testing to detect any vestibular abnormalities.

Results: In 38 patients, there was a positive history of ear fullness, tinnitus and attacks of rotatory vertigo. Hearing was normal in 25 and in 13 there was a sensorineural loss not matching their age and sex norms and unrelated to any other comorbidities. Vestibular testing revealed abnormal HIT in 30 patients, caloric hypofunction in 42 patients, and abnormal c-VEMPs in 28. All patients with audiovestibular symptoms were previously treated before referral with poor response. Patients with abnormal tests were shifted to acetazolamide and had a dramatic response on both audiological and vestibular manifestations.

Conclusion: Changes in CSF pressure significantly affect inner ear fluids in some patients. Symptoms and tests may mimic Meniere's disease and we recommend evaluating patients with atypical MD, especially early bilateral affection and/or poor response to conventional therapy, for increased ICT. Further testing of other cochlea-vestibular functions in these patients is under way.

MENIERE'S DISEASE AND VESTIBULAR MIGRAINE

Marco Mandala
Italy

Both migraine and dizziness/vertigo rank among the most common complaints in the general population. Vestibular migraine is among the most common cause for recurrent spontaneous vertigo with a lifetime-prevalence in the general population of about 1%. Menière's disease is the vestibular disorders that display the most increased prevalence of migraine. Comorbidity between Meniere's disease and vestibular migraine is frequent and their symptoms overlap. Etiology and pathogenesis of the two diseases is at the present time at least in part unknown. The latest theories will be presented with special emphasis on the familiar-genetic background. Diagnostic criteria for the two diseases will be detailed. Outcomes of vestibular test for Meniere's disease and vestibular migraine will be presented. Specific treatments both for the acute attack and prophylaxis of Meniere's disease and vestibular migraine as well as their association will be discussed.

BILATERAL MENIERE'S DISEASE

Ronen Perez
Department of Otolaryngology and Head and Neck Surgery, Shaare Zedek Medical Center affiliated with the Hebrew University Medical School, Jerusalem, Israel

The bilateral variant of Meniere's disease is an extremely challenging entity in terms of diagnosis and treatment. The condition is known to have a significant impact on the patients' quality of life. It's incidence in published reports widely varies and is estimated as being between 2-78% of general Meniere's patients. Shojaku and colleagues from Japan, in a recent large study, reported an incidence ranging from 9-16%. In a study by our group on a 101 patients who underwent intra-tympanic gentamicin treatment, only 5% developed full contralateral disease while a higher percentage of patients had low frequency hearing loss in the contralateral ear (16% at 0.25 and 0.5 kHz and 29% at 0.25 kHz only). Some clinicians believe that bilateral disease presents within 2-5 years of the initial onset but it is our experience that this period is much longer and there is no defined interval beyond which the patient can be considered "safe".

The diagnosis of bilateral Meniere's disease can be difficult. We believe that similarly to unilateral disease the diagnosis is primarily clinical with additional tests being corroborative. Nevertheless, in recent years additional tests have emerged and aid in the diagnosis. Namely, cervical and ocular evoked myogenic evoked potentials (cVEMP and oVEMP). In addition, there are interesting reports on the role of high-resolution gadolinium-enhanced MRI for the diagnosis of Meniere's disease, which can be helpful in the diagnosis of bilateral disease.

There are several clinical differences between unilateral and bilateral Meniere's disease, which affect the patients' quality of life. The patients experience a chronic disequilibrium, probably due to the bilateral vestibular loss, which is initially transient and becomes permanent. Obviously, the bilateral hearing loss significantly decreases their communication skills. An important clinical feature is the high prevalence of associated migraine in bilateral cases in comparison to unilateral. Ruckenstein and colleagues reported that 41% of their bilateral patients had associated migraine in comparison to only 10% with unilateral disease. Studies looking at health related quality of life issues showed that bilateral patients achieved much lower scores than the unilateral patients. The questionnaires show that bilateral patients perceive their dizziness as more disabling. Their increased anxiety is enhanced by the difficulty in treating these patients. Obviously, only non-ablative treatments are possible. To date modalities of treatment include mainly intra-tympanic steroids and the controversial sac surgery. Systemic aminoglycoside therapy is reserved for the severe and intractable cases.

OTOLITH DISFUNCTIONS AT EARLY AND LATE STAGES IN MENIERE'S DISEASE

Eduard Matsnev, Elena Sigaleva
State Research Center of Russia - Institute for Biomedical Problems, Academy sciences of Russia, Moscow, Russia.

Introduction: It is known that sacculus is the second most frequent site for hydrops formation after cochlea, especially at late stages of Meniere's disease (MD) - (Schuknecht, Gulya, 1983; Okuno, Sando, 1987; Young et al., 2003; Yamane et al., 2011). This study represents the analysis of frequency and severity of otolith dysfunction according to MD stage.

Methods: We present the data of 105 patients (67 women and 38 men) at the age from 19 to 71 years (mean age ± SD, 45 ± 26 years) with unilateral MD analyzed trough 9-year period (2005-2014 years). Stages of MD were defined on the basis of American Academy of Otolaryngology-Head and Neck Surgery recommendations (1995). For patient investigation we used tonal audiometry, VNG, bithermal caloric testing (Fitzgerald/Hallpike), vibration test, Tullio-phenomenon and Hennebert symptoms presence, postural balance estimation. VEMP registration was used for sacculus function assessment. The utriculus was estimated by subjective perception of Subjective Visual Vertical (SVV) and Subjective Visual Horizontal (SVH).

Results: I MD stage was revealed in 34 patients (32.3%); II – in 32 (30.4%); III – in 24 (22.8%); IV – in 15(14.2%) patients. The non-significant interrelation (P>0.05) between unilateral weaknessof caloric reaction and a stage of MD was established.

Positive Tullio-phenomenon and Hennebert symptoms were revealed in 7 patients (10.6%) with I and II stages of MD and in 22 patients (53.8%) with III and IV stage of MD. The presence of these phenomenon in combination with VEMP suppression or its absence (especially in patients with late stages of MD) is well coordinated with the hypothesis of sacculus involvement in hydrops process (Okuno, Sando, 1987; Y-Ho Young et al., 2003). It is well known that saccular dilatation during hydrops in MD patients leads to distribution of pressure upon the footplate, enhancing the sensitivity of the saccular macula to loud sound and mechanical pressure on footplate (Fraysse, Alonso, House,1980). Kwee (1976) suggested that sound stimulates the vestibular neuroepithelium by the means of a dilated saccular wall, which is in contact with the footplate.

In 56 from 66 patients with I and II stage of MD (84.8%) were revealed normal p13 and n23 VEMP latencies (11.2±0,42msec è 20.1±2.1msec, respectively). Among patients with III and IV BM stages normal VEMP values were revealed only in 4 from 39 patients (10.2%).

Sacculus dysfunction in patients with I and II stage of MD was revealed in 10 from 66 patients (15.1%), demonstrating the 50% p13 and n23 VEMP amplitude suppression or its absence. Patients with III and IV MD stages demonstrated the same VEMP disturbances, but statistically (P<0.05) more frequent (in 20 from 39 patients) – [58.9%]. A dilated sacculus with an atrophied saccular macula, which was described in histopathologic study of MD (Schuknecht, Gulya, 1983), could be an explanation for depressed VEMPs. The loss of saccular macula associated with collapse of the saccular wall onto the otolithic membrane is thought to be responsible for a depressed-type VEMP (Y-Ho Young et al., 2003).

Utriculus dysfunction (SVV and SVH subjective perception "mistake" more than 1° ±1SD on the affected side) was revealed in 4 (6%) patients with early MD stages and in 12 (30%) patients with late MD stages.

Postural balance investigations ("Sway Star™" Swiss technique - Allum J.J., Adkin, A.L., 2003) carried out in patients with early (n=5), late (n=5) MD stages, Tumarkin otolith crises (n=5), all of

them in comparison with 5 healthy men, demonstrated the advantages of this test for otolith function estimation in MD patients.

BENIGN PAROXYSMAL POSITIONAL VERTIGO ASSOCIATED WITH MENIERE'S DISEASE

Sertac Yetiser

Recurrent vertigo represents a significant disability almost in all societies. Meniere's disease and BPPV are amoung the common causes of recurrent vertigo which account for 7.8 % and 18.3 % outpatient cases, respectively in the literature. There is a tendency for these syndromes to appear in the same patient which leads to longer duration of symptoms, more sessions needed for cure and higher rate of recurrence. The aim of this study is to document the clinical features of BPPV associated with Meniere's disease. A total of 363 patients with BPPV who have been evaluated between 2009- and 2014 during outpatient clinical visit were enrolled in this retrospective study. The medical records of 18 patients with BPPV associated with Meniere's disease were reviewed. All patients had unilateral Meniere's disease (11 in the RE, 7 in the LE). Sixteen of patients with MD had ipsilateral BPPV where BPPV was in the counterlateral side in 2 patients. Fifteen of patients had well-known MD for a long time before the diagnosis of BPPV. However, 3 patients developed MD during BPPV follow-up with sudden and fluctuating hearing loss. Seven patients had LC-BPPV and 11 patients had PC-BPPV. Thirteen patients had BPPV symptoms (positional and brief vertigo) more than 2 months (assuming a delay of diagnosis due to presence of MD). Twelve patients (including all patients with LC-BPPV) required two or more maneuvers for the relief of symptoms. Conclusively, BPPV associated with MD differs from idiopathic BPPV in regard to several epidemiological and clinical features. In view of data from patients with MD and BPPV, possible underlying interrelations were discussed.

PREVALENCE OF SYSTEMIC AUTOIMMUNE DISORDERS AS CO-MORBIDITY IN MD

Suchitra Prasnsuk
Hearing Speech Balance Tinnitus Center, Bangkok Hospital Medical Center, Bangkok

Meniere's disease is common inner ear disorder characterized by episodic vertigo of spinning type associated with hearing loss and tinnitus at the time of attack. Hearing loss is of fluctuated cochlear type. There is still uncertainty on etiology of the disorders and uncertainty of co-morbidities. Autoimmune disorder was considered to be one of co-morbidity but there was rare study on autoimmune disorders in MD patients may be due to expensive test and result is not so cost effective and not so satisfy to clinician. Patients might not agree to have the test since it is expensive and if it is positive it will be a stigma to the health care insurance later on.

Method: Our Bangkok Hospital is a super tertiary care hospital provides service to not only affordable Thai but people around the world. Our hearing and balance center provides high standard of services. We saw many MD patients in a year around 300 visit a year. For Vertigo Academic we select the definite MD came to see us during the pass 6 months with at least two episode of attacks with fluctuate hearing loss of cochlear type. There are 22 in number age 22-72 Male 11: Female 11 which has been sent to immunology dept. of immunological study. First they all need to answer to all questionnaires then follow by blood test for CBC, ESR, VDRL, TPHA, Thyroid function, ANF, ANA, RF, ANCA, TSF,ASO, SLE, anti HBc, anti CCP Ig6, anti HCV select individually.

Result: Positive finding found in 4 cases out of 22 cases which is 18.18 %. Two are SLE + ve, one positive RF and SNSA, another one only RF +ve. Detail will be discussed.

15:30-16:00

MOTION SICKNESS

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Over two thousand years ago the Greek physician Hippocrates wrote, "... sailing on the sea proves that motion disorders the body ...". The word 'Nausea' derives from the Greek root word 'naus', hence 'nautical' meaning a ship. In the modern world, motion sickness can be provoked by many types of transport by land, sea, air and space. Moving visual images can also trigger motion sickness and new visual technologies may put more of the population at risk. The primary signs and symptoms of motion sickness are nausea and vomiting. Related symptoms can include vertigo, dizziness, stomach awareness, sweating and facial pallor ('cold sweating'), increased salivation, sensations of bodily warmth, drowsiness ('sopite syndrome'), headache, loss of appetite and increased sensitivity to odours.

The 'how' of motion sickness (i.e., mechanism) is generally accepted to involve sensory conflict. This sensory conflict is between actual versus expected invariant patterns of vestibular, visual, and kinaesthetic inputs, as predicted by an 'internal model'. Two main types have been proposed: (i) conflict between visual and vestibular inputs or (ii) mismatch between canals and otoliths. The long predicted 'sensory conflict' neurones in the brainstem and vestibular nuclei have been identified and the underlying brain mechanisms are being elucidated. The same brainstem areas seem to mediate vomiting and nausea irrespective of the triggering mechanism, whether by motion or by toxins.

But what purpose does motion sickness serve if any? This is the 'why' of motion sickness, which can be viewed from both evolutionary and non-functional mal-adaptive theoretical perspectives. The most popular is the 'toxin detector' hypothesis which proposes that the brain has evolved to recognise any derangement of expected patterns of vestibular, visual, and kinaesthetic information as evidence of central nervous system malfunction and to initiate vomiting as a defence against a possible ingested neurotoxin. On this basis, motion sickness in pedestrian man or other animals is simply the inadvertent activation of this ancient defence reflex by the sensory conflicts induced by the novel altered visual and force environments of transports by sea, air, land, space and virtual reality.

Individual differences in susceptibility are great. MZ and DZ twin studies indicate heritability for motion sickness susceptibility around 60%, and genome studies have begun to identify multiple contributing genes. Infants and very young children appear unaffected by motion sickness, susceptibility then rises with age to peak at around 9 years of age, declining thereafter into adulthood and older age, perhaps due to habituation. Irrespective of age, females tend to be more susceptible than males, although there is much overlap. Reliable physiological markers for susceptibility have proven elusive. Shorter time constants of the central vestibular velocity store have been suggested to correlate with reduced motion sickness susceptibility. But it may be the ability to modify the time constant easily that may be more important than the absolute time constant. Motion sickness susceptibility varies dramatically between types of patients. Patients with bilateral vestibular loss are almost immune from motion sickness. Patients with vestibular neuritis or BPPV have susceptibilities overall equivalent to normals. Those with vestibular migraine, Meniere's disease and migraineurs show greatly elevated susceptibilities.

Counter-measures can be behavioural or pharmacological and all have relative advantages and disadvantages. Habituation (desensitisation) is the most effective countermeasure but is time consuming and may be stimulus specific. More rapid behavioural approaches include obtaining stable horizon views, avoiding head movements, adjusting body posture, being in control, and controlled breathing. Unfortunately these are often precluded by the particular situation or required work activities. Anti-motion sickness drugs divide into: anti-muscarinics (e.g. scopolamine), H_1 anti-histamines (e.g. dimenhydrinate), and sympathomimetics (e.g. amphetamine). Combinations (e.g. scopolamine+dexamphetamine) are highly effective since both drugs combine their different anti-motion sickness properties, and their respective side-effects of sedation and stimulation cancel each other out. These combinations are no longer available because of risk of abuse and legal reasons. Other classes of drugs have shown no major advantage over those currently available for motion sickness. The reasons include relative lack of efficacy, complex and variable pharmacokinetics, or in those that are effective, unacceptable side-effects. Future developments should aim for highly selective affinities to receptor subtypes relevant to motion sickness, consequently with high efficacy and few side-effects.

16:30-17:00

LECTURE: SUPERIOR CANAL DEHISCENCE

Issam Saliba
Canada

Superior canal dehiscence (SCD) syndrome patient's present with a cochleo-vestibular symptom. Several cochlear symptoms have been reported, with autophony, hyperacusis, symptomatic hearing loss, aural fullness or pressure, and pulsatile tinnitus being the most commonly encountered. Vestibular symptoms, such as imbalance, vertigo, oscillopsia, Tullio phenomenon, and Hennebert sign, are also usually found in a large percentage of patients with SCDS.

Superior canal dehiscence is a benign condition in which a surgical treatment may be considered depending on the patients' tolerance of their symptoms. The natures of cochleovestibular signs and symptoms were shown to be key factors in patients' choice of a surgical management whereas paraclinical tests seem to be less significant in the patients' decision for a surgical treatment.

In our center, all patients diagnosed and surgically treated for SCDS were operated through a middle fossa craniotomy (MFC). Clinical, audiological and radiological data are always collected. Air-bone gaps, Pure-tone average (PTA), speech discrimination scores (SDS) and VEMP thresholds were correlated to dehiscence size.

Air-bone gaps, VEMP and computerized tomography remain essential tools in diagnosing and following SCDS. Dehiscence size is an independent factor in the analysis of SCDS, with cochlear symptomatology being associated to dehiscence sizes. Overall symptomatology, audiometric results and VEMP thresholds return to normal values post-obliteration, confirming the continuing success of the MFC approach for SCDS obliteration.

Nature and number of cochlear symptoms, Valsalva and pneumatic speculum-induced vertigo, VEMP thresholds, and ABGs seem to correlate with a positive HRCT. The ABG at 250 Hz is the most accurate predictor of SCD.

The prevalence of dehiscent-appearing superior canal on thin-section temporal bone scanning with reformation in the SSC plane is much higher than anticipated by pathologic studies. Even with 0.55 mm-collimated helical CT and reformation in the SSC plane, the risk of overdiagnosis is present.

The superior semicircular canal (SCC) can be specifically stimulated in humans using the modified "bilateral simultaneous caloric test" (BSCT). It can provide a good estimation of its stimulation intensity after the surgery.

During the presentation, I will share with the participants a didactic management, an interpretation of all the tests performed when a SCD is suspected, and a movie showing the superior canal plugging through a MCF approach.

17:00-18:00

VESTIBULAR NEURECTOMY WITH SIMULTANEOUS ENDOLYMPHATIC SUBARACHNOID SHUNT

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The purpose of this study was to assess the advantages of combined vestibular neurectomy (VN) and endolymphatic subarachnoid shunt (ELSS) surgeries in classic Menière's disease. We performed a retrospective analysis of the results of 116 patients with classic Menière's disease who were operated on via a posterior fossa approach. All patients underwent selective VN. In 86 of the patients, ELSS surgery was performed in conjunction with VN via the posterior fossa, which is called two-in-one surgery. Among the 86 patients who underwent two-in-one surgery, hearing preservation was achieved in 71 (82.5%), and the vertigo control rate was 96.5%. In patients who underwent VN without ELSS, hearing stabilization was achieved in 24 (80%), and the vertigo control rate was 96.7%. The hearing results and vertigo control

rates were similar in the groups. Aural fullness subsided in 62 (72.1%) of the patients who underwent VN plus ELSS and in 14 (46.7%) who underwent VN alone. The recovery of fullness was significantly better with the combined VN and ELSS procedure (P<0.05). In conclusion, although the two-in-one operation is a new procedure, its results for vertigo control and hearing stabilization are not different from that of VN alone. The only significant advantage of this technique was the achievement of a substantial improvement in the reduction of aural fullness.

SINGLE-SHOT, LOW-DOSE INTRATYMPANIC GENTAMICIN IN MÉNIÈRE DISEASE: ROLE OF VESTIBULAR-EVOKED MYOGENIC POTENTIALS AND CALORIC TEST IN THE PREDICTION OF OUTCOME

Tayfun Kirazlı
Turkey

Objective: The aim of this study was to assess the efficacy and safety of single and low-dose intratympanic gentamicin therapy in patients with Ménière disease and who were monitored both with caloric tests and vestibular-evoked myogenic potentials (VEMPs) to see if VEMPs have an additional role in predicting the efficacy of the drug.

Setting: Tertiary referral center is the study setting.

Patients: Twenty-five intractable Ménière disease patients were included as the study group.

Intervention(s): Low-dose (16 mg/mL), single-shot intratympanic gentamicin was applied. Vestibular-evoked myogenic potential and caloric test were applied 2 weeks after the application.

Main outcome measure(s): Safety and efficacy of protocol were evaluated at the sixth month postoperatively with tonal audiogram and visual analog scale, respectively.

Results: Mean average pure-tone hearing threshold at 0.5, 1, 2, 4, and 8 kHz was 49.6 and 51.0 dB before and after the application, respectively (P N .05). Mean pretreatment and post treatment visual analog scale scores of patients were 17.6 mm (10–30 mm) and 74.6 mm (41–100 mm), respectively (P b .01). Posttreatment VEMPs were absent in 17, deteriorated in 2, and not changed in 6 patients. Vestibular-evoked myogenic potential was a significant predictor of posttreatment visual analog scale score, whereas caloric test was not (P b .01).

Conclusions: Low-dose, single-shot intratympanic gentamicin treatment proved to be effective and safe among intractable Ménière patients. Vestibular-evoked myogenic potentials obtained at posttreatment second week were significant predictors of patients posttreatment sixth-month dizziness status and vertigo control.

EVALUATION OF SURGICAL TREATMENT FOR DISABLING VERTIGO IN UNILATERAL MENIERE'S DISEASE

Franco Trabalzini
Dept of Otology and Skull Base Surgery, Siena University Hospital, Italy

Objectives: We evaluated the effectiveness of different treatment options in patients with disabling vertigo due to unilateral Meniere's disease.

Patienst and Methods: This retrospective study included 169 patients (78 males, 91 females; mean age 50.1 years; range 23 to 80 years) who were treated for recurrent peripheral vertigo from January 1990 to January 2000.

All the patients were classified in accordance with the AAO-HNS (American Academy of Otolaryngology-Head and Neck Surgery) 1995 criteria, with a disability defined in level ≥ 4 of the AAO-HNS Functional Level Scale.

Intratympanic application of gentamicin was used in 29 patients who refused surgery or where surgery was contraindicated. Endolymphatic sac surgery was performed in 20 patients. Labyrinthectomy, either transcanal of transmastoid, was performed in eight patients whose hearing function was absent or not useful in the affected ear. Vestibular nerve section (VNS) was performed in 112 patients who had a good general condition, a serviceable hearing in the diseased ear, a real disability affecting social and professional activities and/or in case of failure of the above mentioned treatments. In the first year, it was performed through the retrolabyrinthine approach (18 patients) and in the latter years through the retrosigmoid approach (94 patients).

Hearing levels were assessed before and 4 to 10 weeks after treatment and vertigo control rates were determined between 18 to 24 months after treatment. The overall follow-up period ranged from 24 to 93 months.

Results: With gentamicin, vertigo improved significantly in 86.2% of the cases (class A 48.3%, class B 37,9%), but some degree of hearing impairment was observed in 41.3%. With endolymphatic sac surgery, deterioration in hearing was 10%; improvement in vertigo control was 65% at the end of a two-year follow-up period. All the patients who underwent labyrinthectomy had vertigo control, with a complete hearing loss. The most beneficial treatment was VNS, in terms of both complete control of vertigo spells (98.3%; class A, 92.9%, class B 5.4%) and preservation of hearing (93.8%). None of the patients in this group required a revision procedure.

Conclusion: As surgical treatment for disabling vertigo in Meniere's disease, VNS is of choice if hearing is worthy of preservation; in patients with unilateral non–useful hearing labyrinthectomy may be considered. In elderly patients and/or in those with a poor health condition, intratympanic gentamicin seems to be the most appropriate option.

SURGICAL OPTION WHEN MÉNIÈRE'S DISEASE OTHER TREATMENTS FAIL

Sofiane Ouhab
Algeria

Introduction: For Meniere's disease a number of strategies may help us manage the symptoms. Our strategy in case ofMeniere's disease resistant to intratympanic applications, mostly If vertigo attacks associated with Meniere's disease are severe and other treatments don't help, surgery may be the option. Procedures we propose are: Endolymphatic sac decompression (ESD), Labyrinthectomy, or Vestibular nerve section.

Materiel and Method: We present a comparative study of different surgical solutions in Meniere's disease.

We performed between 2011 and 2014 in our department in KOUBA public hospital (Algiers, ALGERIA), 17 surgeries for Menière's disease: 4 labyrnthectomy, 6 ESD, 7 vestibular neurotomies. The choice of the procedure was indicated according to the severity of vertigo, hearing status, and choice of patients.

Results: In our experience, patients had better results with the vestibular neurotomy compared to other surgical procedures.

Conclusion: In severe cases, surgery can be performed to control the attacks of vertigo. However, surgical procedures are usually only used if other treatments have failed.

PRESSURE TREATMENT FOR RECALCITRANT MÉNIÈRE'S DISEASE

Maurizio Barbara, Edoardo Covelli
Sapienza University, Sant'Andrea Hospital, Rome, Italy

Despite the lack of significant evidence, all Meniere subjects are addressed to a certain period of medical treatment, consisting of diuretics, low-salt diet and reassurance. It is our personal experience that this regimen would succeed in nearly 70% of the cases, with the rest of the subjects still suffering to various extent of incapacitating spells. Up to 2000, this latter group of subjects has been directly addressed to surgery, consisting of either endolymphatic sac drainage or, more frequently, to vestibular neurectomy. From 2000 on, the advent of a pressure-delivering device has shown to change the evolution of the disease in those subjects who, being recalcitrant to the medical regimen, were either sent or selected for vestibular neurectomy. More than 80 subjects with invalidating vertigo (class D or E) have been treated with the Meniett(R) device (Medtronik, USA). The treatment protocol consisted in 5 automatic sessions performed in 1 month period, after positioning of a short-term ventilation tube. In a last group of treated subjects, the outcome has also been assessed via performance of electrocochleography, other than with history collection and questionnaires. When assessed after at least 24 months from the pressure therapy, more than 65% of the treated subjects showed to shift from class D or E to Class A (predominantly) or B, so as to demonstrate the long-term efficacy of the treatment. Only in few of these cases, the pressure treatment needed to be repeated at a certain distance from the first one.

Considering the potential morbidity of a neurotosurgical procedure, the pressure treatment could be considered a viable therapeutical alternative that showed to be easy to perform under self-administration setting and devoid of complications in respect, for instance, to gentamycin instillation.

SURGERY FOR VERTIGO

Prepageran Narayanan
Malaysia

Dizziness remains a very common presenting symptom in primary care setting. It can be a daunting and frustrating if a proper clinical approach is not used. Detailed approach in a clinical setting ranging from history, clinical examination and investigations are discussed in the presentation. The complete neurotological examination usually tests different components of the vestibular-central system and this includes otological examination, examination of vestibular ocular reflex, cerebellar signs and gait. The following are specific peripheral vestibular tests. Surgical management are usually reserved for recalcitrant cases which fortunately are rare.

BPPV: It is characterized by rotatory vertigo that occurs when the head is rolled to a certain position, such as when turning in bed, getting out of bed or looking upward to reach for an object placed above the head. The condition is caused by free-floating particles in the posterior semicircular canal that moves with head movements. This is confirmed by the Dix Halpike manoeuvre. Treatment consists of a repositioning manoeuvre that moves the debris into the vestibule where it is asymptomatic. This treatment is typically effective in more than 90% of cases and in recalcitrant cases, surgical occlusion of the posterior semicircular canal can be carried out.

Ménière's DiseaseB Ménière's disease is characterized by episodic vertigo, tinnitus, fluctuating sensorineural hearing loss and aural fullness. Treatment includes low salt diet and antidiuretics in early stages with betahistadine (a vasodilator). Refractory cases with unilateral vestibular involvement can be managed with ablative procedures of the labyrinth. In selected cases for hearing preservation, vestibular nerve section can be performed. Recently, intratympanic injection of gentamicin has shown promising results.

Recurrent Vestibulopathy: This is a purely descriptive term for a condition of unknown aetiology in patients who have experienced more than a single attack of vertigo that is characteristic of Ménière's disease but without the auditory signs and symptoms. Patients may present purely with vertigo of minutes to hours without hearing loss, tinnitus or aural pressure. Treatment is usually similar to that of Ménière's, i.e. with betahistadine. Some cases resolve spontaneously while other progress to Ménière's disease.

22 MAY 2015 / WORKSHOP HALL

11:30-12:00

NEW VESTIBULAR AND VHIT DATA CASE STUDIES

Michelle Petrak
USA

With recent improvements in video technology and goggle design, screening vestibular system function through the use of video Head Impulse Testing (vHIT) is now a reality. This presentation will provide an introduction to the Video Head Impulse Test and how it can add value to vestibular practices. The Video Head Impulse Test (vHIT) is a fast, simple and objective measurement for assessing semicircular canal function in each canal independently. A small goggle, high-frequency camera, inertial measurement device and calibration laser are used to capture, record and analyze eye/head movements. vHIT is quickly becoming the new gold standard in vestibular assessment allowing for quick identification of vestibular deficits in any clinical setting. vHIT, combines with VNG and VEMP for a complete vestibular diagnostic test battery. This course will utilize case studies to demonstrate different types of vestibular dysfunction and compensation patterns. It will focus on measurements of instantaneous gain and velocity regression gain. It will explain how and why saccades are triggered in impaired vestibular function and will discuss the stages of compensation. The case studies are intended to relay a greater understanding of vestibular disorders to course participants.

12:00-12:30

DYNAMIC POSTUROGRAPHY: WHERE AND HOW DOES IT CONTRIBUTE?

Michel Lacour
France

Balance performance of healthy subjects as well as of vestibular loss patients can be measured using kinematic or dynamic approaches for the purpose of motion analysis or force quantification, respectively. The dynamic approach used in clinical investigations of posture control is based on stable force platforms, that is, relatively simple and low-cost techniques, and on moving dynamic force platforms which are more complex and expensive tools better adapted to test pathological subjects and to follow the efficacy of a vestibular rehabilitation therapy protocol.

A substantial challenge when investigating posture control is to record signals that are effective in describing static/dynamic balance control and to interpret these signals in terms of postural performance or strategy. The traditional describers of posture control, based on the centre of foot pressure (CoP) displacements (total length path, surface of the stabilogram), lead in many cases to misevaluations of the balance-control system. For instance, decrease in CoP area can attest of postural improvement that results from a better integration of the multisensory inputs controlling posture, or from increased-body stiffness associated with fear of falling.

New describers of posture control open access to more functional aspects of balance performance. Carried out according to fractional Brownian-motion analysis, the stabilogram-diffusion analysis provides the spatio-temporal parameters over which posture control switches from open-loop to closed-loop control mechanisms, or from reliance on sensory inputs to co-activation of antagonistic muscles. The wavelet analysis is another method that can be used to extract more useful information on posture control mechanisms. In contrast to the Fourier analysis, the wavelet transform elaborates a time-frequency chart of body sway and provides a 3D representation of body sway with the frequencies of CoP displacement as a function of time, the third dimension being given by the spectral-power density of body sway, which is a good proxy of the energy spent to control balance. Bode plots (gain and phase) of body sway are available with dynamic posturography platforms using sinusoidal displacements in the anterior-posterior and medio-lateral directions, giving therefore access to the strategy of balance performance. The energy and the time required for body re-stabilization after perturbation of balance by a sudden ramp translation are other useful parameters when translational platforms are used.

Where and how does dynamic posturography contribute to a better understanding of postural performance will be evidenced by comparing these traditional and new describers of posture control mechanisms in different healthy subjects (young versus older adults) and in different pathologies affecting balance control (vestibular loss patients, multiple sclerosis, cerebellar stroke, Parkinson disease, visual dependency). Results will be illustrated also by comparing these different populations examined in different experimental conditions (eyes open, eyes closed, under optokinetic stimulation, with the dual-task paradigm).

12:30-13:00

HOW TO PERFORM AND INTERPRETE THE VHIT

Laurent Tardivet
France

VHIT systems allow to analyse the VOR deficit of each semicircular canals. We present our way to perform this test and point out the hints and pitfalls that the examiner must recognize.

Synapsys software uses several guides to keep only validated impulses and train examiner to generate reproducible head thrust.

The learning curve is a factor to take into account, and determine the accuracy and the quality of the test.

New features divide this test in two parts.

The first one analyses the semicircular canal VOR and the second one the central compensation strategy – the saccade –

Saccades are automatically analysed, and each parameters can be detailled as the apparent gain, the latency and the frequency of their occurence.

We will precise how we can understand the results given by this device, check the reliability of each impulse, and suggest tracks to follow and diagnosis to evoke.

16:30-17:00

INTRATYMPANIC STEROIDS FOR MENIERE DISEASE, PRELIMINARY RESULTS

Sergei Kosyakov, Alexander Gunenkov
Russian Medical Academy of Postgraduate Education, Moscow, Russia

Background: Currently the intratympanic treatment of different diseases of internal ear by steroids is considered to be the most effective and became very popular. Intratympanic administration of steroids, even in small quantities, results in higher concentration of the drug in the end organ, than in the case of systemic administration. Therefore, drugs that are used topically in low doses can be preferred.

Objective: Investigation of the efficiency of intratympanic route of steroid administration in control of vertigo attacks in Meniere patients.

Methods: Up to now 11 patients with proved Meniere diseases after unsuccessful conservative treatment with betagistin and reologic therapy have been included in the study. All the patients have been treated with intratympanic dexamethasone (IT-Dex) over a period of 6 months, according to the protocol designed for the Idiopathic sudden sensorineural hearing loss treatment. Puretone audiograms were compared before and after the treatment, and each vertigo attack was fixed.

Results: Intratympanic therapy showed the treatment efficiency during the 1-year period in 2 cases and more than two years control of vertigo in 9 cases. PTA didn't show any changes.

Conclusions: Treatment intratympanically administered steroids over 6 month according to our protocol demonstrate the efficacy in vertigo control in patients with Meniere disease.

IT therapy can be used as a secondary method for treatment of patients with unsuccessful previous treatment for vertigo control. Further studies are needed.

INTRATYMPANIC (TRANSTYMPANIC) MULTIPLE CORTICOSTEROID APPLICATIONS IN PATIENTS WITH NON SURGICAL THERAPY RESISTANT MENIERE DISEASE

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Introduction: Meniere disease remains an etiological and therapeutically challenge. Even in single sided Meier patients there can be seen difficult cases who are resistant to standard therapy. Patients with bilateral Meier diseases are at risk to lose functional hearing as well to loss ability to independent life. In our medical centre surgical therapy for patients with Meniere disease was never introduced. Also gentamycin as an intratympanic therapy is lately excluded from therapeutical protocol.

Patients and Methods: A serial of patients with Meniere disease, diagnosed and treated in the last three years in our Audiovestibulological centre will be presented. Two patients with resistance to conventional therapy, with recurrent vertigo attacks and fast progression of hearing loss will be presented. In these two cases multiple intratympanic (transtympanic) applications of concentrated Dexamethason (24mg/ml) was used. In one case also the tympanic tube was inserted. In one case hearing loss reached level of hearing loss of medial level and in one case of severe hearing loss. In specific cases the therapeutically approach should be personalized to specific patients who does not respond to classical therapeutically protocol.

Results: In majority of cases more conservative transoral therapy in not so frequent classical attacks in patients with Meniere disease can be successful. In two cases out of all treated patients only multiple intratympanic (transtympanic) corticosteroid therapy was successful in one patients and in the second one was only partial successful.

Conclusion: Even repetition of intratympanal corticosteroid therapy in difficult cases of patients with therapy resistant Menier disease can lead to less favorable end results regarding hearing and vestibular function. The possible other therapeutic modalities will be discussed.

17:00-17:30

ACUTE VESTIBULAR LOSS: MANAGEMENT PART I

Fazıl Necdet Ardıç
Turkey

A patient with acute vestibular loss may attend to the outpatient clinic or emergency service with the complaints of vertigo, dizziness or lightheadedness. The first step for the management is the differential diagnosis. In this presentation the clinical signs and symptoms, evaluation methods are summarized for differential diagnosis.

History taking, examination of vestibulo-oculer and vestibulo-spinal reflexes are explained. Their value for differentiate the diseases are highlighted. Eye movements, head shaking test, head thrust test, Romberg test, unterberger test, past pointing, cerebellar test are shown with videos. The diagnostic algoritms for diagnosis are explained in details.

ACUTE VESTIBULAR LOSS AND THE TREATMENT

Cem Bilgen
Ege University School of Medicine, Izmir, Turkey

Acute vestibular loss is a clinical condition characterized by vertigo or dizziness that develops acutely in seconds, minutes or hours. It is accompanied by nausea and vomiting, ataxia, spontaneous nystagmus and motion intolerance. It generally persists for a few days. The symptoms resolve functionally over a period of 6-8 weeks due central compensation. Mostly, the underlying disease is a benign peripheral vestibular disorder, particularly vestibular neuritis. However, central causes, particularly brainstem and cerebvellar pathologies, can mimic benign peripheral

causes. Whne planning the treatment of a patient with acute vestibular loss, the predictors of a central pathologies should be exclusively evaluated. Exclusion of the central causes indicate the managemnet of the paripheral causes, i.e. vestibular suppression and supportive treatment. In the current session, the treatment of acute vestibular loss and the predicting issues for the central causes will be presented.

17:30-18:00

HOW TO DIAGNOSE AND TREAT BPPV OF ANY CANAL

Daniele Nuti
University of Siena, Italy

Benign paroxysmal positional vertigo (BPPV) is a common syndrome caused in most cases by displaced otoconia floating in the semicircular canals making them gravity sensitive. It is characterized by brief episodes of vertigo, precipitated by rapid change in head position. Because of its anatomical position, the posterior canal (PC) is by far the most frequently involved; less often, otoconial debris enter the lateral canal (LC) or moves from one canal to another. Bilateral labyrinthine involvement is possible, especially after head or whiplash injury. Anterior canal (AC) BPPV is possible but rare, due to the anatomical position of the AC.

PC-BPPV is induced by a rapid transition from the sitting to the head hanging left or right positions (the Dix-Hallpike manoeuvre). The characteristics of the resulting nystagmus are crucial for diagnosis: after a few seconds, the provoking manoeuvre causes a mixed torsional-upbeat, paroxysmal and transitory nystagmus; on return to the upright position there is a reversal of direction. LC-BPPV differs from PC-BPPV mainly in that the vertigo is more intense and principally caused by rotatory movements of the head or body in supine position and in that nystagmus is horizontal instead of vertical-torsional. The specific manoeuvre to detect positional nystagmus due to LC-BPPV is called "Supine head roll test". Depending on the different locations of otoconial debris in the canal, LC-BPPV can be divided into two variants, the more common with geotropic nystagmus and the less common one with apogeotropic nystagmus.

The management of BPPV relies on conservative, physical therapy. The aim of physical therapy is to eliminate the episodes of positional vertigo by making the otoconial debris to come out of the semicircular canals. There are two main types of treatment for PC-BPPV, the Semont liberatory manoeuvre and the canal repositioning manoeuvre proposed by Epley, and its modifications. These are known since late eighties and both are surprisingly effective, with more than 80% of favorable results after one treatment. Both manoeuvres, especially that of Semont, cause a so-called liberatory nystagmus from which the effectiveness of the manoeuvre can be predicted.

There are at least three therapeutic methods that give good results for LC-BPPV: the so-called "barbecue rotation" proposed by Thomas Lempert in 1994, the "forced prolonged position" suggested by Paolo Vannucchi and collegues the same year and the Gufoni's maneuver conceived in 1998. These therapeutic approaches are even very effective in releasing patients from the disease in a short time.

Many physical procedures have been proposed for the treatment of AC-BPPV but at present, no controlled studies are available, and their effectiveness is sometimes questionable.

18:00-18:30

NYSTAGMUS OF CENTRAL ORIGIN

Ossama Mansour, Amr Gouda
Egypt

Different forms of nystagmus could be seen in central lesions, like Upbeat, Downbeat, pendular, gaze-evoked, See-saw, Periodic alternating and Convergence-retraction nystagmus. Nystagmus of central origin are often pendular, do not have a fast-slow phase, and is vertical in direction; even though horizontal and jerk nystagmus can occur with central lesions. A better way to differntiate between peripheral and central nystagmus is by fixing the gaze and see if nystagmus is reduced or relieved. In peripheral nystagmus, it is often relieved by gaze-fixation while central nystagmus is not. Another important differentiating clue is the associative sign and symptoms; including cerebellar sign such as ataxia, dysdiadokinesia, intention tremor, and scanning speech; and brainstem sign such as bulbar palsy, hemiplegia, or unilateral sensory loss. But this may not be enough, central lesion can be cerebellar, brainstem, posterior hemisphere and cerebral hemisphere.

Upbeat nystagmus is due to pontine lesion which result from the damage of ventral tegmental tract (VTT) originating from the superior vestibular nucleus(SVN). This tract course through the ventral pons and transmitting excitatory upward vestibular signals to the 3rd (oculumotor) nerve nucleus. Thus any lesion that disturbed this pathway could result in upbeat nystagmus. In addition, similar nystagmus is produced from lesion of caudal medulla. Interestingly, there is no clinical case of downbeat nystagmus caused by focal lesion of the brainstem. Downbeat nystagmus is usually caused by lesion of cerebellar flocculus, which in turn resulting in disinhibition of SVN-VTT pathway, followed by relative hyperactivity which drive the upward slow-phase. Downbeat nystagmus are seen in structural lesion of the cervicomedullary junction such as Chiari-malformation. Other possible causes include any form of lesion to cerebellar flocculus.

As regard pendular nystagmus, it invariably occurr in total blindness. When the eyes is unable to fix a vision for example in a sudden dark room, there will an attempt to fix in a previously remembered location. If this response mechanism is disrupted, as in the case of lesion to the optic nerve (optic neuritis or multiple sclerosis), there will be pendular nystagmus. This also explains the presence of pendular nystagmus in congenital blindness. However, lesion of the cortico-pontine-cerebellar or olivocerebellar pathway could yield similar pendular nystagmus. Thus showing the hypothesis is incomplete and pendular nystagmus has a wide range of causes.

In gaze-evoked nystagmus, there is also subdivision into vertical and horizontal. Not much is known about its pathophysiology

but is stated that it is due to lesion of the neural integrator function located in the brainstem, specifically nucleus prepositus hypoglossi (at medulla) for horizontal integrator and interstitial nucleus of Cajal (upper midbrain) for vertical. Damaged involving these area will give rise to gaze-evoked nystagmus of respective direction. Similar presentation could occur in corresponding vestibulocerebellar pathway lesion.

See-saw nystagmus is usually associated with parasellar lesions such as pituitary tumour, or any lesion to the optic chiasm. See-saw nystagmus has been reported in visual loss dues to retinitis pigmentosa.

Periodic alternating nystagmus (PAN), presented as horizontal (almost always) nystagmus, then stopped and followed by reverses direction of the nystagmus; this cycle is repeated usually every minute. The presumed mechanism is damage to the vestibulo-ocular tract at the pontomedullary junction; usually a cerebellar lesion or brainstem lesion. Hence, it is also has a wide range of causes.

Convergence-retraction nystagmus, perhaps the most "pathognomonic" of all type of waveform, presented as adducting saccades (medial rectus contraction). This can occurred spontaneously or during an attempted upward gaze (often accompanied by retraction of the eyes into orbits). This typically points toward mesencephalic pretectal region (dorsal midbrain) lesion, usually a compressing pinealoma.

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08:30-09:00

FROM THE EARS TO THE EYES: USING THE BRAIN IN BETWEEN TO SHARPEN OUR DIAGNOSTIC TOOLS

David S. Zee

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The last 25 years has seen extraordinary progress in the laboratory evaluation of the dizzy patient. With vestibular evoked myogenic potentials (VEMPs) we can probe the function of the utricle and saccule. With quantitative video-oculography (VOG) we can probe the functions of individual pairs of coplanar semicircular canals. Without minimizing the importance of these advances, much can still be learned about our patient's problems if we hone our bedside examination skills (after all our visual sense is the best vestibular testing tool we have!) and then interpret our clinical observations using the basic physiology of how the labyrinth senses the position and motion of the head and what the brain does with that information.

Ewald and Flourens taught us that the eyes rotate in the same plane as a stimulated semicircular canal. Hence stimulation of a vertical canal produces a mixed vertical torsional nystagmus. This simple but fundamental principle allows us to better interpret the patterns of nystagmus in patients with benign positional vertigo, semicircular canal dehiscence and selective involvement of the superior vestibular nerve. It also allows us to localize certain patterns of nystagmus as being central such as pure torsional and pure vertical nystagmus. The geometric relationships of the semicircular canals in the head is propagated into central neural networks, including those that generate saccades and quick phases of nystagmus. Hence we have the general principle that pure vertical eye movements require stimulation of both sides of the brain, much as a pure vertical slow phases require stimulation of both anterior (or both posterior) semicircular canals. The optokinetic system and even the pursuit system show similar organizational features, and reflect the importance of the underlying vestibular anlage for all types of conjugate eye movements. Ewald's second (excitation is a better stimulus than inhibition) and third (vertical canals are excited by ampulla fugal motion) laws, too, have immediate clinical impact, e.g., the head impulse sign.

A second fundamental principle relates to the Romberg sign of the vestibulooocular reflex. A hall mark clinical dictum is that nystagmus arising from peripheral lesions is suppressed by fixation while nystagmus that is central in origin is not. The former is probably largely true, but with two caveats. The fixation mechanism can sometimes be overwhelmed with a particularly strong peripheral nystagmus, and the fixation mechanism is best for suppression of horizontal, less for vertical and least for torsional nystagmus. Thus one can be misled as to what the directional vector of a nystagmus is unless one measures the nystagmus without fixation.

The wave form of the nystagmus, is it constant velocity or velocity changing (increasing or decreasing) and how the pattern of nystagmus changes with changes in eye position in the orbit or with convergence can be key localizing features.

The physiology of various patterns of skew deviation including the ocular tilt reaction are best understood and localized in the context of the emergence of phylogenetically-old, static ocular righting reflexes in frontal-eyed animals. Also relying on the otolith organs are translational vestibuloocular reflexes (e.g., side to side or fore and aft) which are most developed in animals with foveas which, in turn, brings a new set of visual requirements for single, binocular stereoscopic vision of small targets. In sum, while the vestibular system is highly complex, careful clinical observation of eye movements, couched in basic physiology, commonly provides the key to a patient's diagnosis.

09:00-10:30

PERILYMPH FISTULA

NAZIM KORKUT

Turkey

One of the most controversial issues in otology is perilymph fistula (PLF). Reason for the controversy is attributed to incomplete understanding of the pathophysiologic mechanism of this disease. Perilymphatic fistula is an abnormal connection between the inner and middle ear. There are a lot of causes of PLF from surgery, trauma, neoplasms and infection (with or without cholesteatoma) or less commonly, it can occur spontaneously due to congenital abnormalities. Such perilymphatic fistulas cause sudden or fluctuating conductive or sensorineural hearing loss, tinnitus, vertigo or dizziness. There is no clear evidence for the existence of spontaneous PLF. There is no method of determining the diagnosis preoperatively; even surgical exploration often depends on subjective interpretation of the findings. Primary treatment of PLF involves conservative measures. Bed rest, head elevation at all times, avoidance of physical straining is advised for 5-10 days. Intractable otological symptoms after this period warrant an exploratory tympanotomy for definitive diagnosis and repair of fistula.

GENETICS OF PERIPHERAL VESTIBULAR DISORDERS

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Vestibular disorders can show different clinical phenotypes, including an episodic vestibular syndrome or a persistent vestibular hypofunction with a progressive clinical course.

Vestibular migraine and motion sickness are the most common episodic vestibular disorders in the general population, after being paroxysmal positional vertigo, and both are related to migraine and show familial clustering. The genes PVRL3, GPD2 and ACO1 are associated with motion sickness. Bilateral vestibular hypofunction is a progressive vestibular syndrome of unknown etiology causing oscillopsia and chronic imbalance. If

the patient also shows abnormal visual enhanced vestibulo-ocular reflex, ataxia and a sensory neuropathy, it is known as CANVAS (cerebellar ataxia, neuropathy and vestibular arreflexia syndrome), a recessive genetic disorder.

However, most patients with vestibular disorders also have sensorineural hearing loss (SNHL), but the cochlear and vestibular symptoms are not temporary associated, being the exception Meniere's disease (MD).

Several non-syndromic deafness also are associated with vestibular symptoms, such as DFNA9, DFN11, DFNA15 and DFNB102, being caused by mutations in COCH, MYO7, POU4F3 and CLIC5 genes, respectively. Furthermore, enlarged vestibular aqueduct syndrome (EVAS) and MD are two endolymph-related disorders that usually manifest with cochlear and vestibular hypofunction. EVAS is caused by mutations in the SLC26A4 gene encoding pendrin. We have identified >90 multicase families with MD and estimated a prevalence of 8.4% of familial MD in a large cohort of Spanish patients with MD. Although we have observed genetic heterogeneity, but most families had an autosomal dominant inheritance with anticipation, and no clinical differences were found between sporadic and familial MD, except for an early onset in familial cases.

By whole-exome sequencing, we have identified two heterozygous single-nucleotide variants in FAM136A and DTNA genes, both in a Spanish family with three affected cases in consecutive generations, highly suggestive of autosomal-dominant inheritance. We have also demonstrated that FAM136A and DTNA proteins are expressed in the neurosensorial epithelium of the rat crista ampullaris. While FAM136A gene encodes a mitochondrial protein with unknown function, DTNA encodes a cytoskeleton-interacting membrane protein involved in the formation and stability of synapses with a crucial role in the permeability of the blood – brain barrier.

By reprogramming lymphocytes from patients with these mutations, we have already generated and characterized an induced pluripotent stem cell model to decipher the role of FAM136A and DTNA genes in MD.

High throughput sequencing, including whole-exome sequencing will be an essential tool for the genetic diagnosis of vestibular disorders leading to a novel taxonomy in otology and neurotology.

Funding: This study was funded by a grant from Meniere's Society, UK

MÉNIÈRE'S-LIKE VESTIBULAR DISORDERS

Natalia Kunelskaya

Russia

Vestibular dysfunction is an important medical and social problem. Every third man over 50 years old experiences dizziness. 10% of ENT doctor's patients and 5% of general practitioner's patients have complaints of dizziness, vertigo and balance disorders. Vestibular disorder is not a disease, but a syndrome, which accompanies various diseases. In patients with vertigo or dizziness the correct diagnosis is extremely important, since from it depend the treatment strategy and therefore the outcome of the disease in the vast majority of cases. Diagnosis of vestibular disorders is complex, because the symptoms are very similar - patients complain of dizziness, vertigo, unsteadiness, gait disorders – but the characteristics of these complaints are different.

A lot of doctors identify the presence of vertigo in patients with Ménière's disease (MD). MD is characterized by attacks of vertigo accompanied by nausea and vomiting, which last several hours and end by itself. During the attack the patient has tinnitus and unilateral fluctuating sensory hearing loss (SHL). The disease develops in young adults (25-40 years) and is associated with the inner ear dysfunction, which is based on idiopathic endolyphatic hydrops.

The same symptoms may present in cochleovestibular disorders associated with vertebrobasilar insufficiency (VBI). However, VBI manifests with dizzinesss or vertigo in patients over 50 years old. The attacks last from few seconds or minutes to several days, can be accompanied by various autonomic manifestations and are often triggered by special factors (head position, emotional stress, fatigue, changing atmospheric pressure, etc.). In addition, patients with VBI have a lot of somatic and neurological complaints: headache, memory impairment, asthenic syndrome, transient changes in visual acuity, including blurred image, diplopia, etc. The diagnosis of VBI is based on such clinical features as the presence of concomitant cerebral and diffused otoneurological symptoms, which demonstrate the involvement of the nuclear zone in medulla oblongata, cognitive and emotional disorders, as well as organic changes in the brain and hemodynamically relevant changes in cerebral vessels, mainly in the vertebrobasilar basin.

The symptoms of benign paroxysmal positional vertigo include paroxysmal vertigo attacks, which are often idiopathic, but always provoked by changes in head position. The attacks last less than 30-40s and are never accompanied by hearing loss and tinnitus.

Vestibular neuritis (VN), as well as MD, occurs most often in young age (30-35 years) and is characterized by severe vertigo with nausea and vomiting. However, vertigo in VN is persistent, strong, dwindling, continues for several days, and sometimes weeks. It's important that there is no hearing disorders.

Labyrinthitis is characterized by severe balance disorders, vertigo with nausea and vomiting, as well as hearing loss (sometimes deafness). Usually it's a complication of suppurative otitis media, sometimes it can be caused by meningitis, viral infection, etc. Vestibular disorders disappear within several months due to central vestibular compensation.

The cardinal symptoms of perylimph fistula is vertigo (persistent or episodic), fluctuating SHL and tinnitus. Most often perylimph fistula is a result of trauma (head trauma, barotrauma, ear trauma, etc.) or physical exercises, which accompanied by pressing.

Minor's syndrome (superior canal dehiscence syndrome) is characterized by vertigo or imbalance with oscillopsia, fluctuating SHL, hyperacusis, tinnitus. This syndrome is triggered by pressing, sneezing or loud noises (Tullio phenomenon). Itcanbeobservedafterconcussionorbarotrauma.

In addition to analyzing of the clinical aspects the diagnosis of the disease, followed by vertigo or dizziness, should be confirmed by laboratory examination: electrocochleography (objective detection of endolymphatic hydrops); video-oculography

(with caloric testing, rotary chair testing, Dix-Hallpike test); vestibular-evoked myogenic potentials (objective testing of saccule and utricle disorder); computed tomography of petrosus bone structures (identifies abnormalities, destruction and traumatic defects of the petrosus bone); magnetic resonance imaging of the brain; ultrasound investigation of brain vessels; audiological examination (diagnosis of hearing loss).

PREDNISONE TREATMENT FOR VESTIBULAR NEURITIS

Avi Shupak[1,2], Anthony Issa[1], Avishay Golz[2,3], Margalit Kaminer[1], Itzhak Braverman[2,4]

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Objective: To evaluate the value of corticosteroids in the treatment of vestibular neuritis (VN).

Design: Prospective controlled randomized.

Methods: Thirty VN patients, 15 in the study and 15 in the control group, were the subjects of the study. The study group was treated by 1 mg/kg prednisone for 5 days, followed by gradually reduced doses of prednisone for an additional 15 days, and vestibular sedatives for symptomatic relief during the first 5 days after presentation. The control group received a placebo and similar vestibular sedatives. The patients had a baseline evaluation and follow-up examinations after 1, 3, 6, and 12 months. The groups were compared for the presence of symptoms and signs, caloric lateralization on the electronystagmography (ENG), the presence of other pathologic findings in the ENG, and Dizziness Handicap Inventory scores.

Results: No differences were found between the groups in the occurrence of symptoms and signs, degree of caloric lateralization, presence of other ENG pathologic findings, and Dizziness Handicap Inventory scores at the end of the study. Complete resolution was observed in 64% of the study and in 80% of the control group. The study group showed earlier recovery of ENG lateralization at the 1- and 3-month follow-up evaluations and higher rates of complete resolution at the 3- and 6-month follow-up points.

Conclusion: Prednisone therapy might enhance earlier recovery but does not improve the long-term prognosis of VN. The clinical and laboratory parameters in VN are not correlated, and both are required for complete patient evaluation.

11:00-11:30

CENTRAL AND PERIPHERAL VESTIBULAR DISORDERS

Mete Kiroglu
Turkey

Peripheric vertigo has the following features:

- Spontaneous, uni-directional, horizontal nystagmus (opposite ear)
- Patients can suppress nystagmus, looking to lesion side, provocation
- Drop towards lesion side
- VOR is pathological (head impulse test)

Central vertigo has the following features:

- dizziness (vertigo),
- diplopia,
- dysarthria,
- dysphagia
- dysmetria (cerebellar ataxia)

For differential diagnosis we have to check the following points during our examination:

Nystagmus

- 1-doesn't change direction
- 2-is horizonto-rotatory
- 3-no skew deviation
- 4-pathological head impulse test

THE DIAGNOSIS OF CENTRAL AND PERIPHERAL VESTIBULAR DISORDERS IN OUTPATIENTS DEPARTMENT

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Introduction: Dizziness ranks among the most common medical complaints in the general population. After headache, it is the second most common complaint of outpatients, not only in neurology. Vertigo is not disease entities, but rather unspecific syndromes consisting of various etiologies and pathogeneses: peripheral vestibular disorders (e.g., benign paroxysmal positional vertigo, BPPV), central vestibular disorders (e.g., stroke), cardio-vascular disorders (e.g., orthostatic arterial hypotension), somatosensory disorders (e.g., polyneuropathy), and others. Most syndromes of vertigo can be correctly diagnosed only by means of a careful medical history and physical examination of the patient (e.g., BPPV). The most frequent disorders causing dizziness, such as BPPV, vestibular neuritis, Meniere's disease and vestibular migraine, are usually accessible to treatment. These effective treatment options for many causes of dizziness justify the need for efficient and reliable diagnoses of outpatients with dizziness complaints.

Materials and methods: We examined referral letters and medical records of 300 patients with dizziness complaints aged 25 to 75 years who were treated in outpatients department of clinic of Nervous Diseases at I.M. Sechenov First Moscow State Medical University during the years 2009-2014. Every patient was examined by neurologistin outpatients department of clinic of Nervous Diseases, who took the medical history andmade the following tests: the head impulse test (Halmagyi test); position of the sample Dix-Hallpike and McClure-Pagnini; Unterberger and Romberg trials; performed orthostatic hypotension, evaluated emotional status according to the hospital anxiety and depression scale (HADS). The HADS consists of 14 questions, all of which were scored on a 0-3 scale. Two scores were calculated: the anxiety score (HADS-A) and the depression score (HADS-D). All the patients underwent videonystagmography andstabilography. We reviewed referral diagnosis to each patient included in this study and compared them with final diagnoses after examinationin outpatients department of clinic of Nervous Diseases.

Results: Vascular encephalopathy was still the most frequent referral diagnosis, occurring in 48% of the patients before examination in outpatients department of clinic of Nervous Diseases.

The second most frequent referral diagnosis was vertebrobasilar insufficiency(30%). The most common final diagnosis after examination in outpatients department of clinic of Nervous Diseases turned to be BPPV (34%). The second most frequent final diagnosis was phobic postural vertigo (22%). The other final diagnoses are: multisensory dizziness (15%), Ménière's disease (7%), migraine-associated vertigo (5%), vestibular neuritis (4%), lacunar infarcts (4%) and other (9%). Most notably BPPV became the most frequent final diagnosis, as it increased from 1% (4 patients) to 34% (103 patients)afterexamination,including Dix-Hallpike test and McClure-Pagnini test.

Discussion: As a result of the research was revealed low levels of diagnosis vestibular disorders in outpatientsdepartment. An examination of outpatients allowed making the correct diagnosis, which is of therapeutic relevance. Most patients with BPPV can successfully be treated with the appropriate repositioning maneuver. The use of the vestibular rehabilitation on the posturographic platform with biofeedback training is an effective method of rehabilitation of patients with multisensory dizziness,Ménière's disease,vestibular neuritis. Most other vestibular disorders are also well treatable if correctly diagnosed.

Key words: dizziness,vestibular disorder, diagnosis

11:30-13:00

MIGRAINE RELATED VERTIGO COVERING DIFFERENTIAL DIAGNOSIS, TREATMENT OPTIONS AND RELATIONSHIP WITH MIGRAINE

Anirban Biswas
Neurotologist, Vertigo and Deafness Clinic, India

Vestibular migraine is a term approved by the International Headache Society (IHS) that is used to describe episodic vertigo in patients with a history of migraine or with other clinical features of migraine. Alternative terms used more or less synonymously include migraine-associated vertigo, migraine-related vestibulopathy, migrainous vertigo, benign recurrent vertigo, and benign paroxysmal vertigo of childhood, highlight some of the uncertainties surrounding our understanding of this disorder. Criteria established by IHS for diagnosing this disorder include episodic vestibular symptoms of variable duration, with at least two of the following in two or more attacks viz –migraine type headaches/ photophobia/ phonophobia/ visual symptoms/ some aura, h/o typical migraine headaches in the past and some documentable vestibular abnormalities in the symptom free period. Though these are set criteria approved by IHS, in clinical neurotological practice we do encounter cases pretty frequently that present with episodic vertigo but do not qualify for all the stringent parameters set by IHS but respond very well to migraine prophylactic therapy especially to drugs like Topiramate, Divalproex Sodium, Propranolol, Amitriptylin etc.. Migraine Associated Vertigo is usually diagnosed from the clinical pattern and by excluding alternatives. Meniere's disease is diagnosed from the criteria laid down by the AAOHNS and vtreated vby set guidelines that include salt restriction, diuretics, and high doses of Betahistine. The principal differential diagnosis for Migraine Associated Vertigo is with Ménière disease. Though the traditional teaching has been that any patient presenting with episodic vertigo of 20 minutes to 12 hours with fluctuating but documentable hearing loss and tinnitus that aggravates during /prior to vertigo attacks is to be suspected as Meniere's disease and episodic vertigo of variable duration without hearing loss but with a history of migraine headaches is to be suspected as MAV, yet current thinking shows that there is much in common between Meniere's disease and Migraine Associated Vertigo and that they probably have a common etiology. Hearing loss can very well be present in Migraine Associated Vertigo and what is very important is that there is a lot of clinical evidence that shows that many patients who present with symptoms very suggestive of Meniere's disease but is refractory to the traditional Meniere's prophylactic treatment do respond very well to prophylactic treatment for Migraine. To cap it all, even Prosper Meniere had in a publication in

Gaz.Med Paris in 1861 had suggested some common patho-physiology between Meniere's disease and

Migraine Related Vertigo. Many findings of current research is now establishing this hypothesis. There is an undeniable overlap of signs and symptoms between Meniere's disease and Migraine associated Vertigo.and many studies have shown a much higher prevalence of migraine in patients of Meniere's disease.There is also a higher prevalence of Meniere's disease in patients of typical Migrraine headaches.

Vestibular Function tests VNG, VHIT, the Subjective Visual Vertical test and the ocular/ saccular VEMP are important diagnostic tests but they only document whether there is a vestivular abnormality or not and do not confirm the nature of the patho-physiology i.e., if it is a Meniere's disease or a Migraine Associated Vertigo. Audiological tests with Glycerol test and ECochG help in suspecting Meniere's disease but imaging studies like MRI has limited value in most if not all cases. Although pathophysiology is as yet uncertain and randomized trials are lacking, treatment recommendations can be made. Some authors have even suggested that it is possible that Endolymphatic Hydrops can develop in an ear that has been previously compromised by the migraine mechanism. Since there is no very typical and foolproof markers for differentiation between Meniere's disease and Migraine Associated Vertigo and a therapeutic trial is often required to differentiate between them, yet it has been suggested that if the duration of episodic vertigo is less than 10-15 minutes / is momentary or has a prolonged duration of a day or more and if audiological abnormalities are less marked in proportion to the clinical vestibular symptoms chances of it being Migraine Associated Vertigo is higher.

MEDICAL TREATMENT OF VESTIBULAR MIGRAINE

Nese Celebisoy
Turkey

Medical treatment of vestibular migraine (VM) involves treatment of individual attacks and prophylactic treatment for patients experiencing frequent, severe attacks interfering with their daily activities.

Attack treatment with migraine specific drugs namely triptans have been studied in two randomized controlled trials. Validity of the first trial with zolmitriptan was limited due to the small

number of patients and attacks though it was superior to placebo in suppressing the attacks (1). In the other study was with rizatriptan the drug was shown to decrease motion sickness after vestibular stimulation in migraineurs (2). The other therapeutic approaches are mainly based on case reports. It is generally accepted to use vestibular suppressants for attacks with prominent vestibular symptoms and to prefer triptans in patients with headache as the main symptom.

Therapeutic studies on prophylaxis are few and they are mainly retrospective cohort studies and open-label trials so the current treatment options are mainly based on migraine guidelines.

In three retrospective cohort studies beta-blockers, valproic acid, topiramate, lamotrigine, clonazepam, amitriptyline and flunarizine have been shown to decrease the duration, intensity and frequency of episodic vertigo and its associated features (3, 4, 5).

In a retrospective, open-label study lamotrigine has been used and was found to decrease the frequency of the vertiginous episodes without a statistically significant effect on headache frequency (6).

In a prospective, open-label study topiramate at 50 and 100mg daily doses has been shown to reduce both the frequency and severity of vertigo and headache attacks (7).

Cinnarizine has been studied in a retrospective, open-label study and the mean frequency of vertigo as well as the mean frequency, duration and intensity of migraine headaches have been found to decrease (8).

In the only randomized control trial with flunarizine the frequency and severity of the vertiginous episodes showed improvement whereas headache frequency and severity did not change (9).

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KEY POINTS OF PATIENT'S HISTORY AND ROLES OF VESTIBULAR TESTS FOR VESTIBULAR MIGRAINE DIAGNOSIS

Onur Çelik

Celal Bayar University Medical Faculty, Otorhinolaryngology Department, Manisa, Turkey.

Migraine related diseases are BPPV, Meniere, vestibular migraine, motion sickness and psychiatric syndrome. Therefore, those are the diseases of differential diagnosis of vestibular migraine. Physicians may have some difficulties to differentiate vestibular migraine especially from Meniere's disease.

Diagnosis of vestibular migraine is made mainly based on patient's history. The key points of patient's history for vestibular migraine are headache and dizziness and/or vertigo.

Characteristics of headache are the nature of pulsatile or pressure type of it. Headache is present most of the patients, but it is not necessary to be simultaneous with vestibular symptoms in vestibular migraine. Headache does not have to exist for the diagnosis. Those patients may complaints other features of migraine like phonophobia, photophobia, and visual aura.

There may be many associate symptoms related to headache like nausea and vomiting, sensitivity to light and/or noise and/or motion, and necessity of going to a dark and quiet place to sleep for relieving the symptoms. Patients mostly describe motion sickness. Almost more than half of those patients have carsickness. The other key point of patient's history is family history of migraine, which is positive more than 50% of the patients. Some cochlear symptoms like ear fullness, tinnitus may be present some of the patients.

Vestibular tests and laboratory investigations has been used for vestibular migraine diagnosis. Vestibular tests such as bi-thermal caloric test, ocular and cervical vestibular evoked myogenic potentials, ocular motor testing, subjective visual vertical, dynamic tilt threshold measurement, video head-impulse test, posturography may show some abnormalities in patients with VM. However, Diagnostic value of vestibular tests is limited; therefore, in clinical practice, diagnosis of vestibular migraine is made clinically rather than tests or investigations. Vestibular tests as well as other investigations like computerized tomography, magnetic resonance imaging, blood tests are may be helpful to exclude the other alternative etiologies.

Keywords: headache; vertigo; dizziness; imbalance; migraine; vestibular; migraine-related vertigo; migraine associate vertigo; vestibular migraine

VESTIBULAR MIGRAINE PANEL

David Zee

Johns Hopkins Hospital, Baltimore, USA

Vestibular migraine is now a recognized clinical entity with specific diagnostic criteria. Its relationship to several abnormal motion perception syndromes is not clear such as mal de debarquement (MDD) and extreme visual motion sensitivity (e.g., flashing strobe lights, excessive motion in the visual environment, etc.). I would like the panel to take up the question about the relationship of vestibular migraine to these common syndromes, including pathophysiology, diagnosis and treatment.

And more specifically what is the relevance of recent ideas on the treatment of MDD including transcranial magnetic stimulation, vestibular adaptation and habituation paradigms (Dai et al) and results of functional imaging implicating specific cerebral cortical areas in MDD (Cha et al.). Are these abnormalities related to abnormally low perceptual thresholds to vestibular stimuli in vestibular migraine (Lewis et al.)?

MIGRAINE AND DIZZINESS

Timothy C. Hain

Professor of Neurology and Physical TherapyNorthwestern University, Michigan, Chicago, USA

Migraine and dizziness are common health problems, and frequently occur in the same person. When symptoms fit within the constraints of the International Headache Society (IHS), the symptoms can be called vestibular migraine. Nevertheless, this labeling of a collection of symptoms does not necessarily mean that the patients have a "disease", and also the labeling process is not particularly helpful to the clinician who specializes in dizziness, whose motivation is to improve the symptoms of his patients, whether or not their symptoms match constraints of the IHS.

In my clinical practice in Chicago, USA, I have treated many thousands of patients with headaches and dizziness, generally attributed to migraine. These patients are largely women in their 50's, rather than the common demographic of migraine in general (women in their mid-thirties). I will discuss results of our algorithm for management, and in particular use of venlafaxine treatment compared to other common migraine medications for prevention.

14:00-14:30

NEUROPHYSIOLOGY OF VESTIBULAR REHABILITATION

Timothy C. Hain

Professor of Neurology and Physical TherapyNorthwestern University, Michigan, Chicago, USA

The vestibular system is a sophisticated human control system. Accurate processing of sensory input about rapid head and postural motion is critical.

Recovery from vestibular lesions has been studied on for over 100 years (Von Bechterew, 1883). Orientation in space and being able to walk upright are critical functions. It is understandable then that the vestibular system is supported by multiple vestibular repair mechanisms. The capability for repair and adaptation is remarkable ! Plasticity consists of neural adjustments that restore original function. This supplemented by substitution of other sensory input or internal estimates. Finally, one may change one's behavior to "work around" problems presented by a vestibular lesion.

Given sufficient time, persons with up to approximately 50% loss of vestibular function adapt so well that a casual observer may find them indistinguishable from someone without a vestibular lesion. Nevertheless, such persons can rarely attain the same degree of performance as normal, and a sophisticated clinician can nearly always detect this situation.

Not surprisingly, the body uses multiple, partially redundant sensory inputs and motor outputs, combined with a very competent central repair capability. The system as a whole can adapt to substantial peripheral vestibular dysfunction.

Our purpose in this overview is to briefly review the neurophysiology of the vestibular system, paying particular attention to aspects relevant to rehabilitation. We will discuss peripheral redundancy as found in the coplanar vestibular canals and pairs of otolith organs, and how it interacts with the "push-pull" wiring that implements common mode rejection in the central vestibular circuitry.

Nonlinear behavior of vestibular afferents may account for saturation phenomena exploited in the "HIT" test. Cerebellar connections are required for compensation. This can lead to severe clinical disorders when there is "double trouble" due to combined cerebellar and vestibular disorders. Somatosensory input, including cervical input is used to substitute for vestibular input in vestibular disorders. It is important to remember that somatosensory input lies in a different coordinate frame than the inner ear, requiring a coordinate rotation to integrate information.

We will conclude with a discussion of "higher-level" problems in vestibular neurophysiology, which are relevant to rehabilitation. The velocity storage mechanism adjusts the timing of responses vestibular afferents to produce outputs closer to desired head velocity input. Velocity storage is generally lost in peripheral vestibular disorders. Internal models, or "Kalman filters" are likely to be the method by which multiple sensory inputs are resolved into a single estimate of body state that is used to drive critical orienting responses. Readjustment of internal models is likely an important function of vestibular rehabilitation.

Overload can drive vestibular afferents into nonlinear behavior – this is exploited by the "HIT" test for unilateral vestibular loss. Ambiguity between tilt and translation, self-movement and world movement commonly can cause imbalance and vertigo. Motion sickness is often provoked by conflicts between sensory streams such as vision and vestibular, or between sensory input and expectations.

Adaptive plasticity for peripheral vestibular lesions is amazingly competent, even enabling the vestibular system to adapt to peculiar sensory situations requiring a reversal of the VOR. (Gonshor and Melvill-Jones 1976) Adjustments of internal models and reweighting of sensory inputs (e.g. Kalman gain, see above) is likely at least as important as readjustment of reflexes, as internal models provide many important features that reflexes cannot provide (such as functioning in the absence of sensory input). The Achilles' heel of the vestibular system is a relative inability to repair central vestibular dysfunction.

14:30-16:00

VESTIBULAR COMPENSATION AND REHABILITATION

Herman Kingma

Maastricht, The Netherlands

Vestibular compensation and rehabilitation are mechanisms to optimise functionality in patients with function loss of the vestibular sense. However, restorage will always be limited compared to the healthy situation, analogue to the fact that lip reading and

hearing aids only partly restore functionality of hearing despite intensive logopedics.

In case of vestibular loss two major issues therefore arise related to treatment. 1. A sudden vestibular asymmetry leads to severe vertigo, nausea and imbalance and 2. loss of speed in image stabilisation during head motion, loss of speed in postural corrections and inadequate vestibular perception of head motion and tilt. Interestingly, there is recent indications from animal research that reduction of the initially symptoms might reduce or delay central compensation. So an interesting point of discussion is: should we reduce the acute vertigo and imbalance by medication at all or is it better to refrain from this approach and focus on optimising central compensation and rehabilitation only to obtain a long term benefit for patients

PANEL ON VESTIBULAR COMPENSATION AND REHABILITATION

Michel Lacour
France

The lecture questions the relationships between the plastic events responsible for the recovery of vestibular function after a unilateral vestibular loss, that is, the vestibular compensation process which has been well described in animal models in the last decades, and the vestibular rehabilitation therapy elaborated on a more empirical basis for vestibular loss patients.

The main objective is to provide clinicians with an understandable view on When and How to perform vestibular rehabilitation, and to explain Why vestibular rehabilitation may benefit from basic knowledge and may influence the recovery process.

With this perspective, 10 major recommendations are proposed as ways to identify an optimal functional recovery rather than to give a catalog of results. Among them are the crucial role of active and early vestibular rehabilitation therapy, coincidental with a post-lesion sensitive period for neuronal network reorganization, and the instructive role that vestibular rehabilitation therapy may play in this functional reorganization when it is performed during this opportunity time window. The need for progression in the vestibular rehabilitation therapy protocols, and the necessity to base these protocols mainly on adaptation processes rather than on habituation exercises are underlined. Why it is important to take into account the sensorimotor and cognitive profile of the patient, and why customized or "à la carte" vestibular rehabilitation should be preferred to standardized protocols are also emphasized. Last but not the least, vestibular rehabilitation therapy protocols should be aimed at reducing anxiety and stress, and motivating the vestibular loss patients. Ecologic contexts can be used as a way to achieve these latter goals.

More than ten general principles are likely, but these principles seem crucial for the fast recovery of vestibular loss patients to ensure good quality of life.

VESTIBULAR COMPENSATION AFTER VESTIBULAR NEUROTOMY

François Caces
Otology and Oto-neurologic Surgery, Causse Ear Clinic, Traverse de Béziers34440 Colombiers, France

Meniere disease (MD) can give birth to invalidant condition, with total disability, when vestibular compensation is not achieved because of persistent fluctuations in the pathologic labyrinth.

Vestibular Compensation is working well when the pathologic disorder of the labyrinth gives stable loss of function.

In Meniere disease, this condition is not achieved, particularly in the early stages of the disease, but can also be seen in later stages when destruction of hair-cells are not advanced.

In a clinical setting, testings to evaluate vestibular activity are: caloric test, VHIT, VEMPs and Subjective vertical. The more frequent test used in the literature is caloric test.

Various questionnaries (Dizziness Handicap Inventory, Meniere Disease outcome Questionnaire, UCLA,..) and American Academy of Otolaryngology-Head and Neck Surgery criteria have been used to assess treatment outcome.

Regarding chance to be totally cured from disability in MD, it could be necessary to consider not only class A and B (AAO-HNS criteriae) but also, disappearance of imbalance and unsteadiness.

It has been demonstrated in several studies that the more hyporeflexic is the treated vestibule, the better is treatment outcome.

Thus, within the limits of our test, it could be expected that areflexy leads to better recovery by the means of improved vestibular compensation.

We present our results in intractable MD patients treated by Vestibular Neurotomy beetwen 2011 and 2013, respecting mean follow-up period of 24 months.

Vestibular neurotomy is the most effcctive way to achieve permanent and stable condition of function because it creates the more complete deafferentation of the pathologic vestibule that we can achieve by treatment.

VOR REHABILITATION WITH HEAD THRUST FOR VESTIBULAR COMPENSATION

Ana Carolina Binetti
Buenos Aires, Argentina

Vestibulo-ocular reflex (VOR) deficits generate a retinal delay which can be perceived as a movement or "Jump" of an object being observed while turning the head. This can be a stimulus for activating cerebellar neuroplasticity through adaptation mechanisms. Moderate to strong evidence shows that vestibular rehabilitation is a safe and efficient treatment for peripheral vestibular disorders. The purpose of this rehabilitation is to decrease sensations of dizziness and the risk of falls as well as to enhance the VOR.

Since the 1990s, the traditional method, called the x1 paradigm, of stimulating adaptation of the VOR has consisted of the performance by the patient of repeated head impulses or thrusts on one plane while at the same time he fixates his vision on a

target (a letter or a point) at a particular distance from the eyes. However, we have observed in our own experience that after carrying out this treatment, evaluations with the video head impulse test (vHIT) showed that there are overt saccades in some patients while in others the VOR gain is insufficient and in still others, both conditions continue to be present. This could be because additional rehabilitation may be necessary for VOR adaptation in which there are high velocity head movements.

For these patients we decided to use a modified vestibular rehabilitation technique for the VOR which was based on a modification of the method published by Migliaccio and Schubert, 2014. These authors did a study on VOR unilateral training with visual incremental stimulus in which they used a scleral coil to measure ocular response in patients who carried out passive and active head thrusts before a moving target. They found gains in VOR adaptation by healthy individuals who did these movements of the head and especially those who did active head movements. In our case, we included patients who after more than three months of vestibular rehabilitation treatment ended up in any one of the three vHIT VOR situations already mentioned and who showed such symptoms of VOR failure as sensations of dizziness when turning the head towards the affected site, the sensation that the eyes "came lately" to the head while moving it, or the sensation of a heavy head or a floating head.

The treatment consisted of repeated passive head thrusts towards the side where the lesion was, which we felt would be predictive for the patient. We did not stimulate the healthy side since we felt it could promote a gain augmentation in the healthy VOR (Migliaccio et al.), with presumed secondary "iatrogenic" dizziness. Stimulus was repeated on the affected side, in 15-20° excursions of the head, with velocities ranging from 150-200°/sec (done under vHIT control). Patients looked at a fixed point target in front of them while doing the treatment. They were evaluated before and after the whole treatment with the vHIT Otometrics ICS Impulse Test; the clinical dynamic visual acuity test; a validated Argentine version of the Dizziness Handicap Inventory (DHI); and the modified clinical test of sensorial interaction and balance (mCTSIB).

In a recent pilot test of controls and patients using the same stimulus, Migliaccio et al. found an increase in VOR gain after treatment, but one which was not statistically significant. In evaluations of the results of rehabilitation with our technique, however, we have observed statistically significant differences in VOR gain asymmetry, in the dizziness handicap inventory, and in dynamic visual acuity in horizontal plane for all patients. We have also found that two patients could not perform the treatment because they experienced increased dizziness while doing so.

We think this could be a promising technique and recommend that it be tested in a larger population.

16:30-17:00

EXPECTATIONS FOR THE FUTURE OF VESTIBULAR DISORDER DIAGNOSIS AND TREATMENT

Charles C. Della Santina

Vestibular NeuroEngineering Lab, Departments of Otolaryngology – Head & Neck Surgery and Biomedical EngineeringJohns Hopkins School of Medicine, USA

The past two decades have witnessed a proliferation of basic, translational and clinical research directed toward improving the diagnosis and treatment of vestibular disorders. Practical results of this research that directly impact clinical outcomes already include improvements in clinical exam maneuvers, diagnostic algorithms, objective tests of semicircular canal and otolith end organ function, genetic tests, systemic and intratympanic pharmacologic treatments, and surgical procedures such as canal plugging for correction of superior canal dehiscence. On the near horizon are vestibular and cochlear/vestibular implants, gene therapy to restore hair cell function, significantly enhanced rehabilitation techniques, and wide availability of relatively inexpensive and portable systems for posture/gait analysis and for binocular 3D video-oculography during highly repeatable canal-specific head rotations. In this lecture, I will review these recent developments and then discuss expectations for the near- and long-term future of vestibular disorder management.

23 MAY 2015 / WORKSHOP HALL

11:30-12:00

A NEW ERA IN THE TREATMENT OF MENIERE'S DISEASE:THE ENDOLYMPHATIC DUCT BLOCKAGE

Issam Saliba

INTERPRETATIONOF VNG

Bernard Cohen
Paris, France

The videonystagmography is an essential and indispensable tool for etiologic diagnosis of vertigo.

Exploration of vertigo has developed enormously in recent years with videonystagmography, which has become richer and more complex. At the same time, it is no longer the only investigation of vertigo. We can study other systems as otolithic system and postural system. We can also highlight with a simple videonystagmoscopy most BPPV.

The videonystagmography was reduced caloric test and oculomotor for years. It is now enriched by rotatory tests increasingly sophisticated, the study of the vertical and the subjective visual horizontal, and the video head impulse test (VHIT).

It allows you to find the arguments

To objectify dizzy with the highlight of a pathological nystagmus

To differentiate peripheral or central aetiology through the study of eye movements, eye fixation index, the search for nystagmus gaze, and the caloric test

Assessing the importance of a vestibular deficit, locate the frequency band of the deficit through caloric test, the rotary testing and VHIT

- Evaluate the vestibular compensation

Participate in the assessment of functional disability

Results:

A / Oculomotricity
Saccade hypometrique
Saccadic pursuit
Internuclear ophthalmoplegia

B / Spontaneous nystagmus or revealed by Head Shaking-Test, bone vibrator,
positional, caloric, rotatory test:
- Intensity> 1-2 ° / s
- Meaning: right or left asymmetry of the vestibular system

C / Isolated directional preponderance or not
Intensity> 1-2 ° / s
Nystagmus spontaneous or latent
Rotatory test
Caloric test

D / Peripheral lesion
Unilateral weakness in caloric
BPPV
Halmagyi-VHIT Test

E / Central lesion
eye fixation index <50%
Gaze Nystagmus
Isolated directional preponderance
Positional nystagmus non BPPV
Internuclear ophthalmoplegia
Skew deviation
Halmagyi-VHIT Test

F / sensitive tests to compensation
Rotatory tests, especially the midrange
Head Shaking Test

G / Non-sensitive tests or partially sensitive to compensation
Caloric test (unilateral weakness)
Halmagyi (HIT; VHIT)

H / Functional exploration of balance through VNG
Study of the optokinetic system with look and stare
Rotation angle estimation

12:00-12:30

MENIERE DISEASE – NEW HORIZONS IN DIAGNOSIS

Paul Avan, Idir Djennaoui, Fabrice Giraudet, Thierry Mom
Neurosensory Biophysics, UMR INSERM, School of Medicine, University of Auvergne, France

Background: The responses of cochlear hair cells to sound stimuli are highly sensitive to the environment and resting position (operating point) of their stereocilia bundles, which need to be adjusted for optimal operation. Among the conditions whereby stereocilia operation is likely disrupted, cochlear hydrops, assumed to be a hallmark of Menière's disease (MD), has the best acknowledged clinical consequences. For several decades, objective diagnosis of endocochlear hydrops has rested upon electrocochleography, as the increased size of the summating potential SP relative to the compound action potential of the cochlear nerve AP is thought to reflect an exaggerated depolarization of hair cells in relation to a deformed, inflated scala media. More recent attempts, which will be reviewed in this presentation, suggest that otoacoustic-emission changes when symptoms of MD are present signal a biased cochlear outer-hair-cell operation, likely in relation to hydrops.

Methods: A cohort of 73 patients with definite MD were enrolled at different stages of their condition, SP/AP ratio and otoacoustic emissions being measured almost simultaneously. The time course of otoacoustic emission (OAE) changes, particularly the phase of OAEs around 1 kHz, and of the SP/AP ratio were followed during body tilt, from upright to supine postures and back. In every tested ear, OAE phase and SP/AP were compared to normative data of the effect of body tilt collected in healthy ears. The sensitivity and specificity of OAEs and electrocochleography were assessed with reference to the clinical status serving as gold standard.

Results: The OAE phase shift with body tilt was excessive in many patients near an attack and still experiencing symptoms of MD, while it went back to the normative interval in most asymptomatic patients. The sensitivity of the OAE test was 75%, and its specificity, 91%. It was noteworthy that the OAE phase displayed

fast fluctuations, within seconds, from normal to excessive values and back. The SP/AP ratio was excessive in less symptomatic patients than the OAE test, with a sensitivity of 60% only. Its specificity was 94%. SP fluctuations, from normal to excessive values and back, were conspicuous in many ears, particularly just after body tilt, on a time scale of minutes limited by the technical constraint of averaging, necessary to obtain reliable electrocochleographic recordings. When combined, the OAE and SP tests reached a sensitivity of 94% without losing specificity (88%).

Conclusion: The use of OAEs and electrocochleography might enrich the study of the cochlear function in patients with hydropic ears, in relation to their symptoms, thereby improving their diagnosis and possibly, the choice of a therapeutic strategy. Fast and slow fluctuations of cochlear function were detected by both tests, only in symptomatic ears, and may reflect the temporal dynamics of cochlear homeostasis.

Acknowledgments: ANR Tecsan Nimopic 2014-2017; Groupe Entendre; Fondation de l'Avenir ET1-617.

12:30-13:00

CEREBELLAR INFARCTIONS MIMICK ACUTE PERIPHERAL VERTIGO

Maxim Zamergrad, Vladimir Parfenov, Ludmila Antonenko

Acute vertigo is a frequent symptom in an emergency department. The main challenge for the neurologist in such cases is to differentiate acute peripheral and central vestibular disorders. This problem can be especially difficult in cases of isolated vertigo. The main and most frequent cause of acute spontaneous isolated vertigo is a vestibular neuritis, which is benign and usually self-resolving condition. However, in some cases such vertigo can be a symptom of central vestibular lesions, for instance stroke or, rarely, multiple sclerosis.

The sensitivity of MRI in visualizing small ischemic lesions in brainstem and cerebellum is apparently quite low. It was demonstrated recently that up to 20% of ischemic lesions can be unnoticed in the first few hours of stroke. On the contrary, it has been proposed that clinical evaluation of the patient with acute spontaneous vertigo effectively recognizes cerebellar strokes and can be more sensitive than MRI.

Strokes with vertigo as an only symptom is usually take place in cerebral regions supplied by vertebrobasilar arterial system. There are two main brain areas, which damage can predominantly provoke isolated vertigo. The first area is in the brainstem near the root entry zone of vestibular nerve. This part of the brainstem is supplied by anterior inferior cerebellar artery. Strokes in the brainstem usually correspond with multiple focal neurologic symptoms among which vertigo can be most prominent. Meanwhile there are few descriptions in the literature of strokes in the area of root entry zone of eight cranial nerve with vertigo as an only symptom. Differentiation of such strokes from peripheral vestibular disorders is extremely difficult and apparently impossible without thorough MRI evaluation.

More often isolated vertigo of central origin can be due to cerebellar lesions. There are two areas in cerebellum the damage of which is responsible for isolated vertigo: nodulus and flocculus. Both of them are usually supplied by posterior inferior cerebellar artery.

Recently it was demonstrated that clinical evaluation of patient with isolated vertigo due to cerebellar stroke can be extremely informative for differentiation of central and peripheral lesions. Such evaluation consists of assessment of spontaneous and gaze-evoked nystagmus, head-impulse test and test of skew deviation. It has been shown that sensitivity of such protocol in diagnosing of cerebellar stroke is almost 100%.

Interestingly hemispheric strokes can also provoke isolated spontaneous vertigo mimicking acute peripheral vestibular lesions. It has been demonstrated that isolated vertigo can be a symptom of the lesions in the vicinity of insular lobe, in parietal and temporal regions and even in posterior limb of internal capsule. These facts can shed light on the distribution of central (especially cortical) vestibular projections.

14:00-14:30

BEDSIDE EXAMINATION

Ali Ozdek
Turkey

In this session bedside examination of patients with vertigo will be discussed. Evaluation of spontaneous and gaze nystagmus and positional tests, examination and evaluation of vestibulo-ocular reflex and vestibulo-spinal reflexes will be discussed. An algorithm will be presented to evaluate dizzy patient.

BEDSIDE EXAMINATION

Bulent Satar
Turkey

In this presentation, bedside examination tools will be mentioned. Bedside examination mainly includes examination of vestibulo-ocular reflex (VOR) and vestibulo-spinal reflex (VSR). Examination of VOR consists of static position of eyes and head, spontaneous nystagmus, gaze nystagmus, gross oculomotor examination, head thrust test, head-shaking nystagmus, positional/positioning nystagmus, visual suppression of VOR, head heave test, fistula test, hyperventilation induced nystagmus, dynamic visual acuity, subjective visual vertical, vibration induced nystagmus. Examination of VSR includes Romberg test, Fukuda's stepping test, walking and tandem gait walking. Importance of these test and clinical examples will be emphasized

14:30-15:00

VERTICAL NYSTAGMUS – WHAT DOES IT MEAN?

Ji-Soo Kim
Department of Neurology, Seoul National University College of Medicine, Seoul National University Bundang Hospital, Korea

Downbeat Nystagmus

Downbeat nystagmus occurs in association with a broad range of disorders, many of which affect the cerebellum or its inputs. Patients with downbeat nystagmus often complain of

oscillopsia, postural instability and clumsiness. Downbeat nystagmus is usually present with the eyes in central position, but often becomes evident only in lateral gaze. Since horizontal gaze-evoked nystagmus is frequently associated, the nystagmus is directed laterally and downward (i.e., diagonally; "side-pocket"). In most patients with downbeat nystagmus, the slow-phase velocity (and nystagmus intensity) becomes greatest in down gaze and least in up gaze (Alexander's law). In some patients, however, downbeat nystagmus is greatest on up gaze. In these cases, the slow phases may not be linear but are, instead, increasing in velocity suggesting an instability of the vertical gaze-holding network. This pattern of downbeat nystagmus has also been observed following removal of the vestibulocerebellum (flocculus and paraflocculus) in monkeys.Convergence often influences downbeat nystagmus, increasing it or sometimes converting it to upbeat nystagmus. Downbeat nystagmus is often precipitated or increased when patients are placed in either a "head-hanging" or prone position. Occasional patients will switch from downbeat to upbeat when they lie supine.When the vertical vestibulo-ocular reflex is tested in patients with downbeat nystagmus using rotational head impulses, at least some patients show an asymmetry with greater responses for downward head rotations than can be explained by addition of nystagmus slow phases to the vestibular eye movements.Such findings suggest an asymmetric sensitivity of anterior versus posterior canal influences in some patients with downbeat nystagmus. Furthermore, following impulsive horizontal head rotations, a downward corrective saccade occurs because the vestibular response also had an (inappropriate) upward slow phase component. Vertical smooth pursuit is impaired for downward tracking in patients with downbeat nystagmus. This asymmetry may be greater than can be accounted for by superposition of the upward slow phases of downbeat nystagmus upon the ongoing pursuit movement.Normal subjects may develop downbeat nystagmus after repetitive upward pursuit, suggesting that pursuit asymmetry may contribute to the pathogenesis of downbeat nystagmus.

Upbeat Nystagmus

Upbeat nystagmus is another type of central vestibular nystagmus that is usually transient and is less common than downbeat nystagmus. Upbeat nystagmus usually increases in upward gaze and accompanies an impaired upward pursuit. Unlike downbeat nystagmus, upbeat nystagmus usually do not increase in lateral gaze, and may change into downbeat during convergence. Upbeat nystagmus has been reported in patients with infarctions, hemorrhages, tumors, multiple sclerosis, Wernicke encephalopathy, epilepsy, brainstem encephalitis, Creutzfeldt-Jakob disease, Behcet syndrome, meningitis, Chiari malformation, and cerebellar degeneration. Transient or paroxysmal upbeat nystagmus may be found in individuals experiencing tobacco, barbiturate, and organophosphate intoxication, and after discontinuation of amitriptyline.

Upbeat nystagmus occurs in association with lesions involving various locations, but most frequently in pontomedullary and pontomesencehalic lesions. It has also been reported in patients with lesions involving the anterior vermis of the cerebellum. In contrast to downbeat nystagmus, paramedian brainstem lesions are frequently observed in upbeat nystagmus.Several mechanisms have been proposed for upbeat nystagmus: (1) imbalance in the vertical vestibulo-ocular pathways, (2) dysfunction of the neural integrator involved in vertical-gaze holding, and (3) impairment of the upward smooth pursuit. However, the exact mechanisms remain to be elucidated.

15:00-15:30

PEDIATRIC COCHLEAR IMPLANTATION AND HORIZONTAL SEMICIRCULAR CANAL FUNCTION

Antonio della Volpe, Antonietta De Lucia

Otology and Cochlear Implant Unit, Santobono_Pausilipon Children' s Hospital of Naples-Italy

Goal of the study: Unilateral or bilateral cochlear implantation (CI) is a safe procedure in experienced hands allowing hearing rehabilitation of patients with severe to profound sensorineural hearing loss. Even though the surgical technique was the same, our pediatric patients aged 7 to 16 years, more in females, reported vertigo or balance problems after CI surgery. In our study we evaluated the influence of CI surgery on horizontal semicircular canal function to test the correlation with symptomatic vertigo.

Material and methods: This prospective clinical study was carried out at our Referral Regional Center for Pediatric CI at the Santobono-Pausilipon Children' s Hospital of Naples-Italy, between 2012 and 2014.

A total of 23 patients, aged 7 to 16 years, undergoing cochlear implantation were assessed pre- and postoperatively for caloric horizontal semicircular canal response and postoperatively for vertigo symptoms.

Results: After cochlear implantation 14 patients had an impaired vestibular function with vertigo symptoms and 8 had a decreased caloric response without vertigo symptoms.

Conclusion: Minimally invasive surgical techniques are important to preserve not only the residual hearing function, but also the vestibular function, even if we have no criteria (age, sex, implant type, petrous bone CT) to identify the risk of postoperatively vertigo.

15:30-16:00

Wiener Vacher (France)

POSTER PRESENTATIONS

PUB NUMBER	PRESENTER	COUNTRY
PP01	**Angelique Van Ombergen**	*Belgium*
PP02	**Angelique Van Ombergen**	*Belgium*
PP03	**Hyo Jung Kim**	*Korea*
PP04	**Floris Wuyts**	*Belgium*
PP05	**Lilian Felipe**	*The Netherlands*
PP06	**Leen Maes**	*Belgium*
PP07	**Leen Maes**	*Belgium*
PP08	**Berina Ihtijarevic**	*Belgium*
PP09	**Berina Ihtijarevic**	*Belgium*
PP10	**Anita Bhandari**	*India*
PP11	**Ercan Kaya**	*Turkey*
PP12	**Natalia Boyko**	*Russia*
PP13	**Natalia Boyko**	*Russia*
PP14	**Alexander Barulin**	*Russia*
PP15	**Diana Demidenko**	*Russia*
PP16	**Alexandra Guseva**	*Russia*
PP17	**Ileok Jung**	*South Korea*
PP18	**Iryna Maryenko**	*Belarus*
PP19	**Avinash Bijlani**	*India*
PP20	**Mustafa Deniz Yılmaz**	*Turkey*
PP21	**Cathérine Blaivie**	*Belgium*
PP22	**Sergei Likhachov**	*Belarus*
PP23	**Olga Kurushina**	*Russia*
PP24	**Mario Milkov**	*Bulgaria*
PP25	**Alexandra Guseva**	*Russia*
PP26	**Chetana Shivadas Naik**	*India*
PP27	**Süheyl Haytoğlu**	*Turkey*

Vestibular Migraine

PP01

VESTIBULAR MIGRAINE IN AN OTOLARYNGOLOGY CLINIC: PREVALENCE, ASSOCIATED SYMPTOMS, AND PROPHYLACTIC MEDICATION EFFECTIVENESS

Angelique Van Ombergen[1], Vincent Van Rompaey[2], Paul H. Van De Heyning[2], Floris L. Wuyts[1]

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Objective: To assess the prevalence of vestibular migraine (VM) in patients consulting to an otolaryngology clinic, the neuro-otological associated symptoms, and the effect of prophylactic antimigrainous medication on VM symptom improvement.

Study Design: Retrospective chart review.

Setting: Tertiary referral otolaryngology clinic.

Subjects and Methods: We used the diagnostic criteria from the Bárány Society and the International Headache Society to allocate patients to a subgroup: VM, possible VM, and atypical VM.

Main Outcome Measure: The prevalence of VM, percentages of associated neurological symptoms (Fig 1), and percentages of effectiveness of prophylactic medication.

Results: Sixty-five (16%) patients were selected from the total patient population (n = 407) from which 4.2% were assigned to the definite VM group, 5.7% to the probable VM group, and 6.1% to the atypical VM group (Fig 2). We found a significantly different distribution between the groups for photophobia (p = 0.035), ear pressure (p = 0.023), and scotoma (p = 0.015). Thirty patients were administered with flunarizine and 68% responded with an improvement in VM symptoms (p < 0.001). For propranolol, 31 patients were treated and there was an improvement of symptoms in 73% (p < 0.001) (Fig 3).

Remarkable was the fact that these percentages were not significantly different between the subgroups.

Conclusion: VM is a common disorder presenting in a dizziness clinic, and detailed history taking is important to assess VM-associated symptoms and thus to prevent underdiagnosis. The latter is very important because our study shows that the majority of patients, regardless of VM subtype, can benefit from a prophylactic treatment, but further prospective studies are necessary.

Keywords: Dizziness, Headache, Migraine, Vertigo, Vestibular migraine

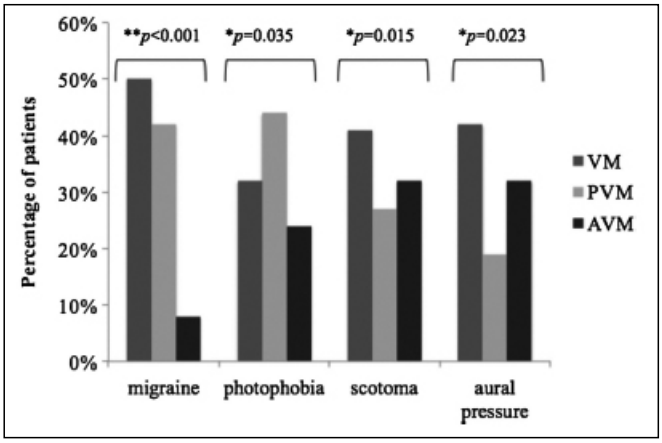

Figure 1. Distribution of symptoms within the different VM diagnostic subgroups.

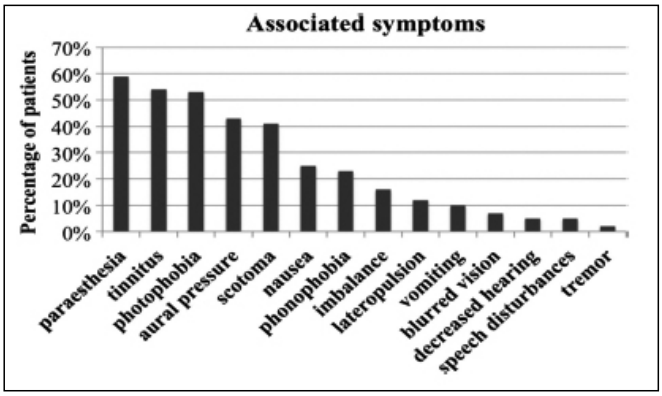

Figure 2. Frequency of various associated neurotological symptoms.

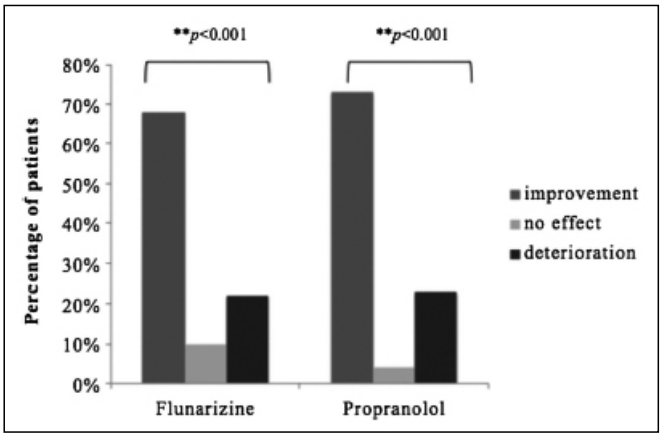

Figure 3. The effectiveness of prophylactic medication on VM symptoms.

Imaging

PP02

REGIONAL WHITE AND GRAY MATTER DIFFERENCES BETWEEN VISUAL VERTIGO PATIENTS AND HEALTHY CONTROLS: PRELIMINARY RESULTS

Angelique Van Ombergen[1], Ben Jeurissen[2], Vincent Van Rompaey[3], Stefanie Vanhecke[3], Floris Vanhevel[4], Paul H. Van De Heyning[3], Paul M. Parizel[4], Floris L. Wuyts[1]

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Introduction and aim: Visual vertigo (VV) is a complex syndrome where patients experience severe dizziness due to disorienting visual environments (e.g. supermarket aisles, crossroads). This could be due to a mismatch at specific brain regions where the integration of visual, vestibular and proprioceptive signals takes place. The aim of this pilot study was to gain insight in the process of mismatch or hampered neuroplasticity in VV patients. We compared patients with a healthy group and investigated differences in the cerebellar and visual pathways in the brain.

Materials and methods: For this pilot study, five VV patients (1 male, mean age: 48.4years) and five healthy control subject (1 male, mean age: 51.1 years) were included. Multi-shell high angular resolution diffusion weighted (DW) data were acquired on a 3T MRI scanner using a 32-channel head coil for all participants. Diffusion tensor imaging (DTI) data were analyzed by means of tractography. In this pilot study, we focused on cerebellar and visuospatial pathways such as cerebellar peduncles, inferior fronto-orbital fasciculus (IFOF) and inferior longitudinal fasciculus (ILF). Voxel-based morphometry (VBM) was also performed to analyze whole-brain gray matter.

Results: We found statistically significant lower values in diffusion measures for the VV patients in the visuospatial network, but statistically significant higher values for the cerebellar network. Furthermore, VBM analysis showed a significant gray matter decrease in these patients in cortical areas known to be involved in the processing of complex visual information.

Conclusions: To our knowledge, this is the first study to use diffusion imaging methods in this specific subgroup of vestibular patients. We found a neurosensory mismatch that could explain the visual vertigo symptoms and the over reliance on visual cues in these patients. This holds a significant clinical importance since this shows a link between subjective symptoms and objective measurements.

Keywords: Visual vertigo, MRI, Diffusion tensor imaging (DTI), voxel-based morphometry (VBM), vestibular disorders

Vestibular Schwannoma

PP03

BILATERALLY ABNORMAL HEAD IMPULSE TESTS INDICATE LARGE CEREBELLOPONTINE ANGLE TUMORS

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Objective: To correlate tumor size with the abnormalities of head impulse tests (HIT) in patients with unilateral cerebellopontine angle (CPA) tumors.

Methods: Twenty patients (14 women, mean age=61±12) with a unilateral CPA tumor underwent a recording of HIT for all six semicircular canals using a magnetic search coil technique. Tumors were graded according to the Hanover classification. Then, the patients were divided into the non-compressing (T1~T3) and compressing (T4) groups according to the grading.

Results: Most (16/20, 80%) patients showed abnormal HITs for at least one semicircular canal (SCC). Furthermore, the HITs were abnormal in both directions in most (7/8, 88%) patients of the compressing group (T4). In contrast, only 25% of the patients in the non-compressing group (T1~T3) showed abnormal HITs bilaterally. Thus, the bilateral abnormalities of HITs were significantly higher in the compressing than in the non-compressing group (chi-square test, p=0.006).

Conclusion: Bilaterally abnormal HITs indicate a larger tumor compressing the cerebellum and brainstem in patients with a unilateral CPA tumor. The abnormal HITs in the contralesional direction may be explained by dysfunction of the flocculus or the vestibular nucleus due to compression by the tumor.

Keywords: Vertigo, Vestibulo-ocular reflex, Cerebellopontine angle tumor, Head impulse test, Flocculus

How I Do It Sessions

PP04

GUIDANCE AND DIFFERENTIAL DIAGNOSIS TOOL OF THE DIZZY PATIENT: "SO STONED"

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We propose a tool that guides the history taking of a dizzy patient. To differentiate the possible aetiologies of vertigo, history taking is of utmost importance but it is not always approached as a checklist.

Several key factors allow a first approximation of diagnose identification and some of them are based on the time profile, symptom profile and trigger profile of the disease. To establish these key factors in a checklist, we propose a mnemonic "SO STONED" that comprises eight different questions that

characterise the vertigo-related complaints of the patient and guide the clinician in his or her decision scheme.

All the letters "SO STONED" have a specific meaning: Symptoms, Often (Frequency), Since, Trigger, Otology, Neurology, Evolution, Duration. Combining all these 8 dimensions of the complaints allow for a discrimination between several diagnoses of vertigo. Each suspected diagnose should be verified by the appropriate clinical and laboratory tests as well as imaging techniques if needed. However, non of the most frequent occurring diagnoses have an identical set of answers to these eight questions of the SO STONED anamnestic checklist. It prooves therefore to be an important tool in the diagnostic approach of the dizzy patient.

S: SYMPTOMS "What are the symptoms? What do you experience as a problem?"

Vertigo, dizziness, unsteadiness, falls, faintness, rotatory or linear sensations, lightheadedness, diplopia, oscillopsia, tilt of the vertical,.

O: OFTEN "How often does 'it' happen?"

Daily, weekly, monthly, irregularly, continuously, only once

S: SINCE "Since when do you suffer from this problem?"

One month ago, a year ago, a decade ago. After a flue, a head injury, a medical intervention, a journey on a boat, train or plane, without any clear cause

T: TRIGGER "What triggers the vertigo or what makes it worse?"

General head movements, walking, rolling over in bed, bending over, taking something out of a closet above your head, driving a car, being a passenger in a car or plane, walking in the supermarket, walking in semi-darkness, or completely spontaneous

O: OTOLOGY "Do you experience any concomitant otological symptoms and when do these occur?"

Hearing loss, tinnitus, aural fullness, hyperacousis.

During the attacks, in between, long lasting

N: NEUROLOGY Do you experience any concomitant neurological symptoms?"

Headaches, migraine, face or limb paraesthesia, scotoma, numbness, palpitations, hyperventilation, speech problems, phonophobia, photophobia

E: EVOLUTION "What is the evolution of the symptoms?"

Status quo, improvement, worsening, ups and downs

D: DURATION "How long lasts the effective vertigo attack?"

Seconds, minutes, hours, days, continuous

Values of effectiveness will be presented to corroborate the usefulness of the SO STONED checklist.

Keywords: Diagnostic-approach, history-taking, mnemonic-tool

Meniere's Disease

PP05

ASSOCIATION BETWEEN VESTIBULAR EVOKED MYOGENIC POTENTIOAL (VEMP) AND HEARING LOSS DEGREE IN PATIENTS WITH MÉNIERE DISEASE

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Introduction: Ménière's disease affects more often the cochlea and saccule. Some studies have shown the potential value of Vestibular Evoked Myogenic Potential (VEMP) in the evaluation of saccular function. The objective of this study is to correlate findings of VEMP testing to hearing loss in patients with established diagnosis for unilateral Ménière's disease. Materials-Methods: Adults of both sexes diagnosed with Meniere's disease were selected to perform the VEMP in an acoustically treated environment. The equipment used was Bio-Logic Systems, Mundelein IL, USA. The acoustic tone burts stimulus (118 dBHL) was monaurally and randomly presented by the computer. The band-pass filter of 10 Hz to 1500 Hz was used. 200 stimuli were presented at a frequency of 1000 Hz. The analysis window was 60ms.The subjects were compared to responses by ear. Results: Among the 18 subjects evaluated, 11 were women. The main change found was the p13 latency increase (38.8%). The hearing loss degree influenced the number of alterations in VEMP. A higher frequency of abnormal response was observed regarding to moderate and severe sensorineural hearing loss. Conclusion: VEMP was confirmed as important evaluation test of Meniere's disease, considering the degree of hearing loss.

Keywords: Meniere disease, vestibular potential, hearing loss

Pediatric Vertigo

PP06

ASSOCIATION BETWEEN VESTIBULAR FUNCTION AND MOTOR PERFORMANCE IN HEARING IMPAIRED CHILDREN.

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Objectives: The literature indicates that 20 to 85 % of children with sensorineural hearing loss demonstrate some type of vestibular dysfunction. Because adequate balance control requires tuning and integration of different sensory input systems, such as the vestibular system, it is reasonable to assume that there is a

connection between vestibular function and motor performance. Therefore, the current study aimed to identify the association between vestibular findings and the motor performance by comparing the clinical balance performance of normal hearing children with those of hearing impaired children with and without vestibular dysfunction.

Methods and materials: Thirty-six children (19 girls, 17 boys, mean age 7y5m, range: 3y8m-12y11m) were divided in 3 different groups. The first group (6 girls, 6 boys, mean age 7y5m) consisted of normal hearing children with normal vestibular responses; a second group (6 girls, 6 boys, mean age 7y6m) entailed hearing impaired children with normal vestibular responses, whereas the third group (7 girls, 5 boys, mean age 7y5m) comprised hearing impaired children with abnormal vestibular function. The two groups of hearing impaired children had a bilateral sensorineural hearing loss and were selected so to that the mean auditory thresholds as well as the distribution of degree of hearing loss, etiology of hearing loss and type of hearing device was comparable for both groups.

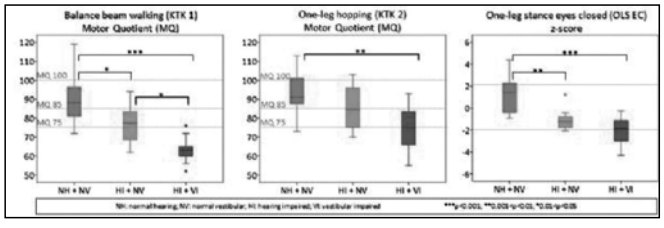
All children were examined with a vestibular test protocol consisting of three sinusoidal rotational tests (0.01 Hz; 0.05 Hz; 0.1 Hz at 50°/s) and collic vestibular evoked myogenic potential (cVEMP) measurements in combination with three clinical balance tests (balance beam walking, one-leg hopping, one-leg stance).

Results: Normal hearing children with normal vestibular responses demonstrated normal balance performance. Hearing impaired children with abnormal vestibular test results, obtained the lowest quotients of motor performance which were significantly lower compared to the normal hearing group ($p<0.001$ for balance beam walking and one-leg stance; $p<0.05$ for one-leg hopping). The balance performance of the hearing impaired group with normal vestibular responses was better in comparison with the vestibular impaired group, but still significantly lower compared to the normal hearing group ($p<=0.01$ for balance beam walking and one-leg stance; not significant for one-leg hopping).

Conclusion: These results indicate an association between vestibular function and motor performance in hearing impaired children, with a more distinct motor deterioration if a vestibular impairment is superimposed to the auditory dysfunction. These findings underscore the importance of vestibular and motor testing in hearing impaired children in order to start appropriate rehabilitation programs at an early age.

Keywords: hearing impaired children, vestibular function, rotatory test, cVEMP, motor performance

Association vestibular function - motor performance



All children were examined with a vestibular test protocol consisting of three rotational tests (0.01Hz, 0.05Hz, 0.1Hz at 50°/s) and cVEMP measurements in combination with three clinical balance tests. The Balance beam walking (KTK 1) and One-Leg hopping (KTK 2) examine dynamic balance ability, whereas the One-leg Stance with Eyes Closed (OLS EC) is a clinical assessment tool for the evaluation of static balance.

PP07

VESTIBULAR FUNCTION AND MOTOR PERFORMANCE IN CHILDREN INFECTED WITH CONGENITAL CYTOMEGALOVIRUS (CCMV) INFECTION.

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Objectives: Cytomegalovirus infection is the leading cause of congenital sensorineural hearing loss, and the prevalence of hearing impairment in children with congenital cytomegalovirus infection (cCMV) has been discussed frequently over the years. Several studies have shown that hearing impaired children are at risk for vestibular damage and delayed motor development. However, the impact of cCMV on the vestibular and motor function has not been investigated thoroughly. Therefore, the purpose of the present study was to evaluate the vestibular function and motor performance in a group of infants infected with cCMV. Furthermore, a comparison of the motor performance was made between children born with cCMV and children born with Connexine 26.

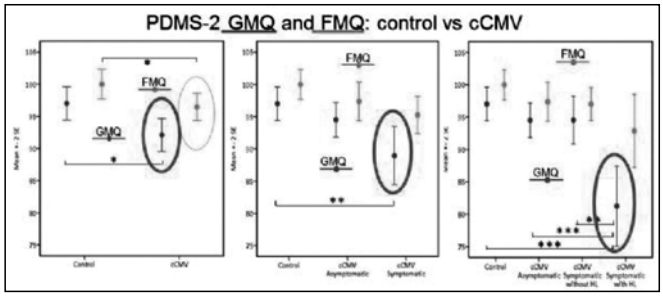
Methods and materials: The motor skills of 45 cCMV-infected children (26 asymptomatic and 19 symptomatic) (mean age 7 months; range: 4 – 13 months) and 45 healthy control children (mean age 8 months; range: 4 – 13 months) were assessed with the Peabody Developmental Motor Scales-2 (PDMS-2). In 22 of these cCMV-infected children (13 asymptomatic and 9 symptomatic) cervical vestibular evoked myogenic potential (cVEMP) testing was performed. The motor performance by means of the PDMS-2 was also compared between 8 children diagnosed with Cx26, 8 symptomatic cCMV-infected children with SNHL, 8 symptomatic normal hearing cCMV-infected children, 8 asymptomatic cCMV-infected children, and 8 healthy control subjects.

Results: Gross ($p=0.012$) as well as fine ($p=0.026$) motor performance were significantly weaker in the cCMV group compared to the control children. Within the group of cCMV-infected children, gross motor performance was significantly weaker in the symptomatic group compared to the asymptomatic children ($p=0.021$). Within the symptomatic cCMV-infected group, hearing impaired children demonstrated significantly weaker gross motor performance in comparison to the normal hearing symptomatic group ($p=0.001$). Absent cVEMP responses were found in 50% of the symptomatic hearing impaired cCMV-infected children, in comparison to a 10% absence rate in the normal hearing symptomatic group and a 0% absence rate in the asymptomatic group. Children diagnosed with Cx26 displayed a better motor performance in comparison with the hearing impaired symptomatic cCMV-infected children, although this difference was not significant.

Conclusion: In general, cCMV-infected children demonstrate a weaker motor performance when compared to healthy children. Moreover, the weakest motor performance and the highest cVEMP absence rate were found in the hearing impaired symptomatic cCMV-infected children. These findings emphasize the importance of early detection of vestibular and motor problems in children diagnosed with cCMV, with special attention to those children also presenting with hearing disabilities.

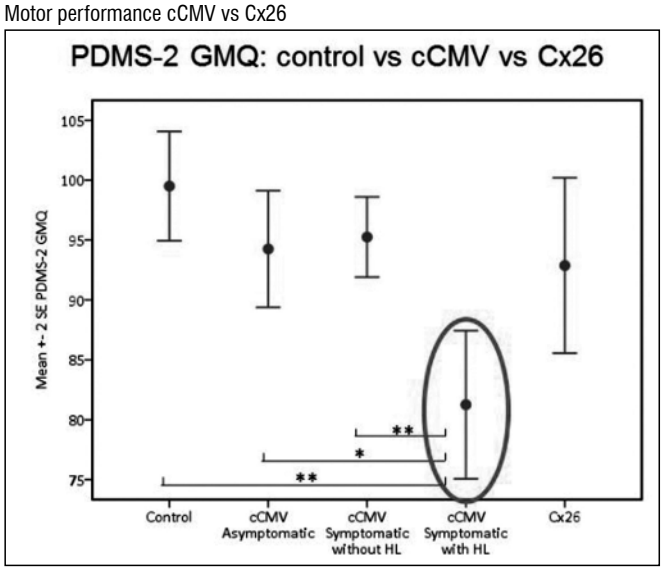
Keywords: congenital cytomegalovirus (cCMV) infection, hearing impaired children, vestibular function, cVEMP, motor performance

Motor performance cCMV



Gross (GMQ) and Fine (FMQ) Motor Quotients were examined by means of the Peabody Developmental Motor Scales - Second Edition (PDMS-2)

Motor performance cCMV vs Cx26



Gross Motor Quotients (GMQ) were examined by means of the Peabody Developmental Motor Scales - Second Edition (PDMS-2)

Vestibular Diagnosis-New tools

PP08

EMBALANCE: A DIAGNOSTIC TOOL TO TREAT PATIENTS WITH BALANCE DISORDERS IN AN EARLIER PHASE OF ILLNESS

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Introduction and Aim: EMBalance is an European Commissioned project led by scientists, engineers and physicians from 7 different countries, including the University of Antwerp. The aim is to design a decision support system (DSS) to aid the evaluation and management of patients with balance disorders.

One of the specific goals is to assess the frequency of different vestibular pathologies among patients presenting at a neurology or ear-nose-throat vertigo consultation. Moreover, we want to identify associated symptoms as well as results from clinical and laboratory testing that occur with different vestibular pathologies. We also want to evaluate the effects of medication and/or other therapy that was prescribed to prevent/treat symptoms. The ultimate goal is to develop a decision support system and to improve the evaluation and management of patients with balance disorders. The system will be based on a database developed by four European clinical centers (UK, DE, GR, BE) where patients with vertigo are seen.

Material-Methods: Data of 1000 patients were collected in different clinical centres. After thorough quality control different groups of patients with BPPV, vestibular migraine, Meniere's disease, vestibular paroxysmia, bilateral areflexia of the vestibular organs, proprioceptive cervicogenic vertigo, chronic subjective dizziness and dehiscence of the superior semicircular canal will be studied. The DSS will be trained using balance-related data. Clinical trials are expected to commence in May 2015 to assess the effectiveness of the DSS.

Results: During the conference, the first results of the Antwerp clinical center will be presented, since database quality control and datamining procedures are still on-going.

Conclusions: Eventually the DSS will form a basis for care pathway for patients with balance disorders.

Keywords: EMBalance, decision support system, vertigo, dizziness, diagnosis, treatment.

Vestibular Paroxysmia

PP09

IMPROVING DIAGNOSTICS IN PATIENTS WITH VESTIBULAR PAROXYSMIA

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Introduction and Aim: Vestibular paroxysmia (VP) is defined as short vertiginous spells frequently dependent on head position with an underlying hypothesis that there is a neurovascular conflict (NVC) with the VIII cranial nerve. Although the syndrome was first described more than 30 years ago by Jannetta, there is still a need for more specific diagnostic criteria to make an early diagnosis and treat patients in an early stage of illness.

Material-Methods: A study was performed on 18 patients with complaints of vertigo, a NVC on MRI and improvement of symptoms by treatment with carbamazepine or oxcarbazepine. Anamnestic and clinical results were examined retrospectively to determine significant similarities among these patients.

Results: In this study 55 % of the VP patients complained of lightheadedness, while 44 % experienced dizziness and 39 % were instable. A smaller group of 33 % experienced rotational sensations as well as a drunken feeling. The most common accompanied symptoms were cervical pain (78 %), hearing loss (55%), tinnitus (61%), headache (55%) and aural fullness (38%). The symptoms occurred daily in 81 % of the patients lasting seconds or minutes (71%) often described by the patient as a continuous burden with short episodes of vertiginous spells. The symptoms were triggered by positional changes (78%), specifically by head movement in 50% and only 27 % by rolling over in bed. Clinical examination showed a persistent non-BPPV type nystagmus (71 %). The Chavda Classification was used to specify the anatomy of the NVC. There was no correlation found between audiometry, electronystagmography and the type of loop. Several other diagnoses were made which led to a mean duration of 16 months and a maximum of 60 months before efficient treatment for VP.

Conclusions: A combination of anamnestic criteria, clinical examination and imaging is necessary to diagnose VP and more importantly to differentiate it easily from other vestibular pathologies as Meniere's disease, BPPV and vestibular migraine.

Keywords: vestibular paroxysmia, neurovascular conflict, vertigo, diagnosis, treatment

How I Do It Sessions

PP10

INNOVATION IN CRANIOCORPOGRAPHY

Anita Bhandari

Vertigo and Ear Clinic

Craniocorpography is a test to evaluate the vestibulospinal reflex function.It involves photographic recording of gait during Romberg's test,Tandem walking and Unterburger/Fukuda stepping test.For this we have devised a special helmet with LED lights to track patient's movement during the tests.Our software gives information about longitudinal displacement,sway,body axis spin and angular deviation with just a click of the mouse.This makes this testing easy,quick,reliable,easily repeatable and gives a hard document for patient records.This helps in evaluation of peripheral and central vestibular disorders and their followup results.

Keywords: craniocorpography, vestibulospinal reflex, sway, spin

Case of right peripheral vestibulopathy

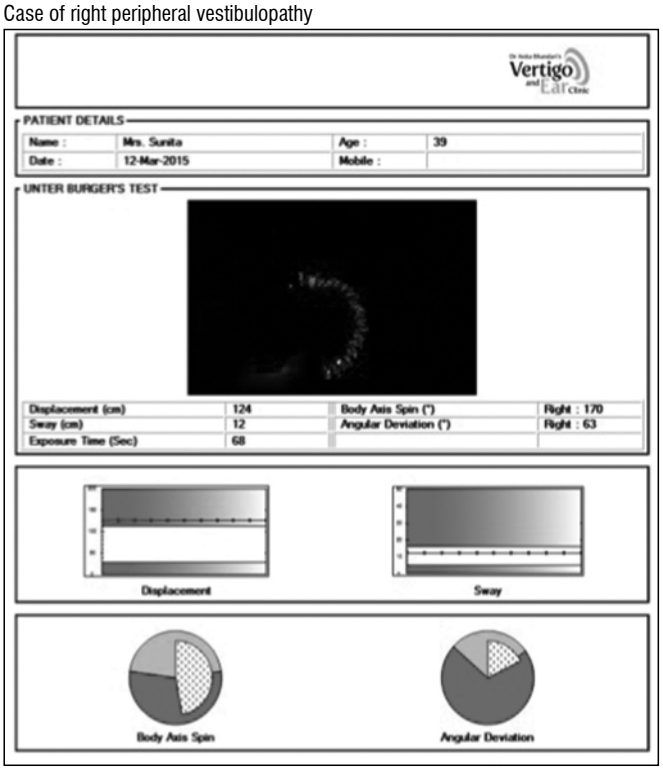

Figure 1. Body axis spin of 170 degrees

Case of right peripheral vestibulopathy

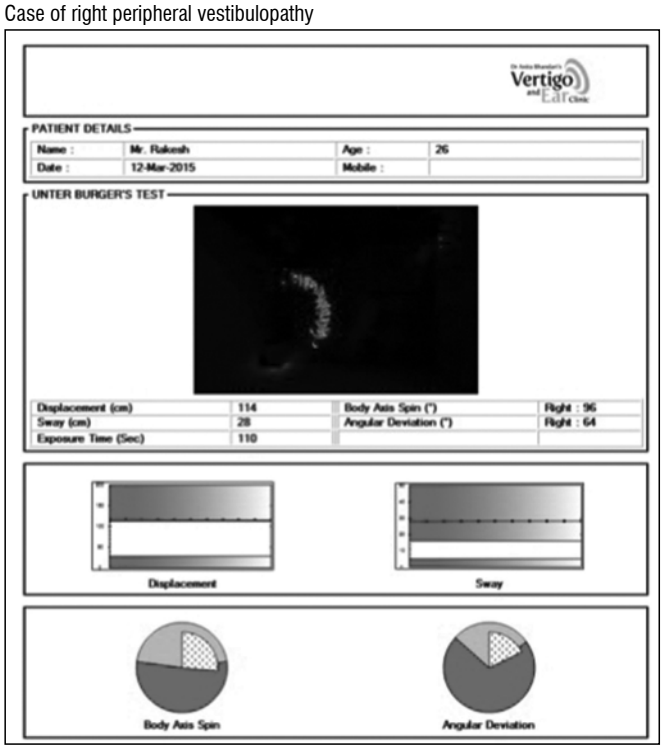

Figure 2. CCG findings of patient showing body axis spin to right 96 degree

Central vestibulopathy

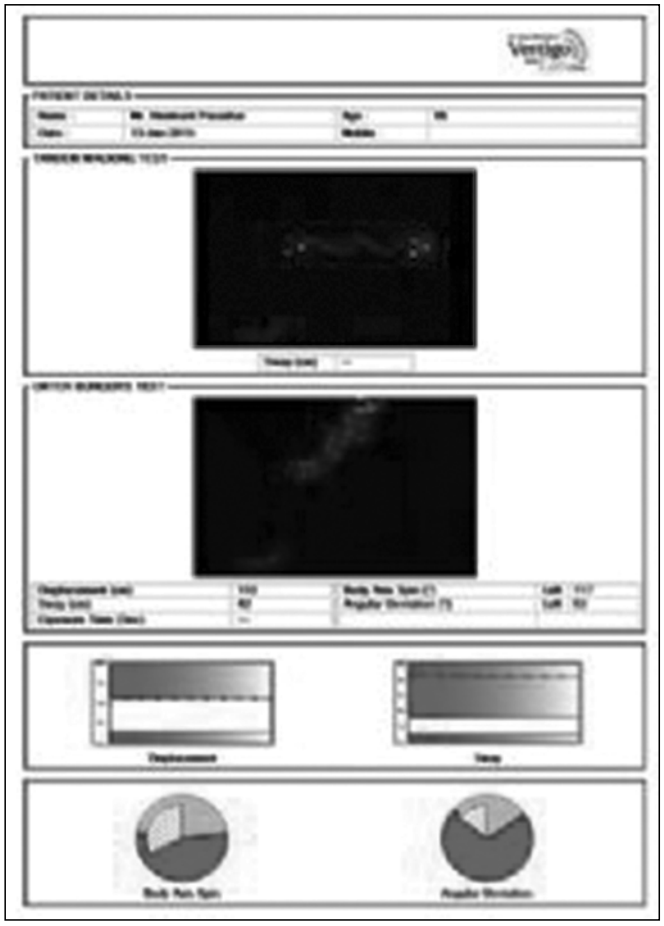

Figure 3. increased sway of 42 cm

Specially Designed Helmet

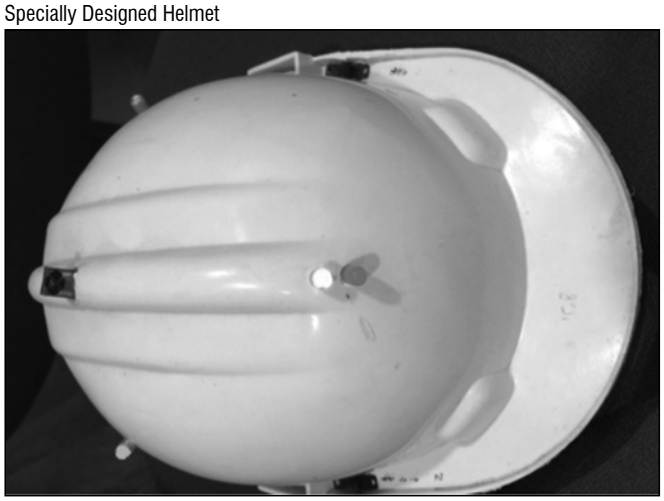

Figure 4. Specially designed helmet with LED lights

Table 1. Normal Parameters of CCG

Parameters	*Normal range Lower border*	*Normal range Upper Border*
Longitudinal Displacement	30.03 cm	113.35 cm
Lateral Sway	5.17 cm	16.15 cm
Angular Deviation	55.13 degree (right)	48.37 (left)
Body Spin	82.21 degree right	82.89 (left)

Table 2. Interpretation of CCG

PATHOLOGY	*CCG FINDINGS*
Peripheral vestibular lesions	Ipsilateral Deviation
Brainstem lesion	Enlarged lateral sway, no angular deviation
CPA tumors, PICA synd.	Contralateral Deviation, enlarged sway

Acute Vestibular Disorders

PP11

DIZZINESS AND VERTIGO AT EMERGENCY ROOM IN GERIATRIC PATIENTS: A RETROSPECTIVE ANALYSIS

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Dizziness and vertigo affect one in five people over the age of 65 years and is associated with substantial healthcare costs. These symptoms indicating severe condition occasionally, are common presenting symptoms in emergency room for elderly.

In this retrospective study, medical data of 552 patients visiting the Emergency Department in Eskisehir Osmangazi University Hospital between January 2014 and December 2014 were analyzed. Demographic parameters, possible diagnosis, treatment, hospitalisation, referral to any department and health-care costs were evaluated. This population included 330(59.7%) women and 222(40.3%) men.

Our analysis showed that in geriatric population vertigo and dizziness are serious symptoms. This symptoms may be first signs of important diseases. In such patients it is necessary to approach multidisciplinary.

Keywords: Dizziness, vertigo, emergency deparment

Perilymph Fistula

PP12

SURGICAL MANAGEMENT OF LABYRINTHINE FISTULA IN CHRONIC OTITIS MEDIA

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Rostov State Medical University

Objective: To analyze clinical presentation, preoperative diagnostic methods and postoperative outcomes of labyrinthine fistula (LF) in patients with chronic otitis media.

Materials and methods: This retrospective study examined the clinical records of 264 patients suffering from chronic otitis media with and without cholesteatoma, who underwent surgical treatment between 2010 and 2013. The age range was 4 to 68 years with an average of 37.2 years. Preoperative examinations included bone conduction audiometry and computed tomography (CT) scanning of the temporal bone. Pneumatic otoscopy was performed in all patients. The fistula test was considered positive if conjugate deviation of the eyes and subjective dizziness were induced by alternating pressure applied to the external auditory canal.

Results: 12 labyrinthine fistulae were found, only 1 was in a patient with chronic otitis media without cholesteatoma. LF prevalence was 4.5%. The fistula was detected radiologically in 10 of 12 patients, whereas two were found incidentally during surgery. 2 of 12 patients revealed labyrinthine fistulae occurring as a late complication of middle ear surgery using the canal wall down technique (Fig. 1). Most patients (10 in number) had the fistula localized in the lateral semicircular canal, while 1 patient had it in the posterius semicircular canal (Fig. 2, 3), and 1 other revealed multiple fistulae (lateral and posterius semicircular canals).

Severity of vestibular disorders did not correlate with LF size and duration of the disease. The results of a fistula test conducted with a Politzer's bulb were positive in 6 patients and negative in 4 patients. In the remaining 2 ears, pressure loading of the ear canal induced the sensation of vertigo without accompanying nystagmus.

To provide complete cholesteatoma ablation, all the patients underwent canal wall down procedure, 7 of them with tympanoplasty. In all cases, total matrix removal was performed, and the fistula covered with bone chops from the mastoid cortical layer, with a free periosteal flap laid upon it.

All the patients were treated with postoperative steroids and antibiotics for 7 days.

The surgery effectiveness criteria were audibility threshold integrity in bone conduction, post-operation fistula symptom disappearance, restoration of integrity of the semicircular duct wall (ac. to computer tomography) (Fig. 4, 5). Postoperatively, hearing remained in the same range in 11 patients. In 1 patient hearing threshold deterioration was observed. Vertigo improved or disappeared in 11 cases.

Discussion: The fistula test does not have a predictive value in preoperative diagnosis of LF, with false-negative rates approximately as high as 50%. The range of true positive results varies approximately from 20% to 70%. In our study, this finding was positive in 8 of 12 cases, which is coherent with the data presented by other authors (Durko M., Durko T., 2003; Copeland BJ, Buchman CA., 2003). Preoperative CT imaging is a more reliable indicator for the assessment of LF.

Conclusion: There is a possibility of labyrinthine fistula in all patients with chronic otitis media. Preoperative diagnosis of the LF is not always possible. Labyrinthine fistula could be present in spite of a negative CT examination.

Keywords: labyrinthine fistula, cholesteatoma, chronic otitis media

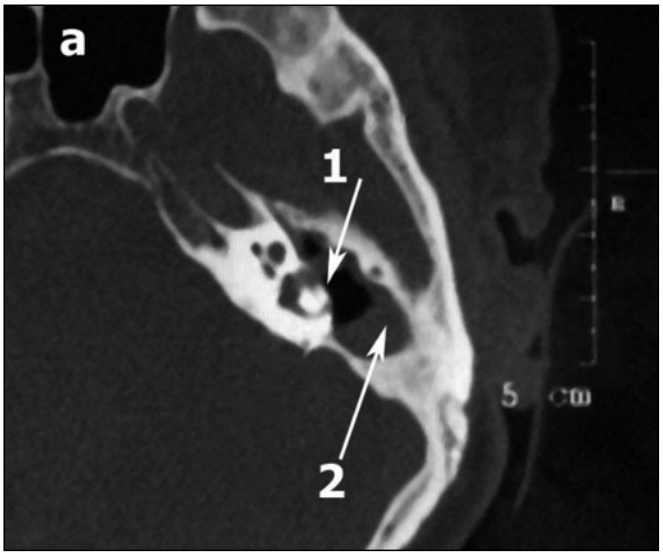

Figure 1. Recurrent cholesteatoma status after canal-wall-down mastoidectomy. Axial CT image demonstrates the fistula of the lateral semicircular canal *(arrow 1)*. Soft tissue fills the mastoidectomy bowl *(arrow 2)*.

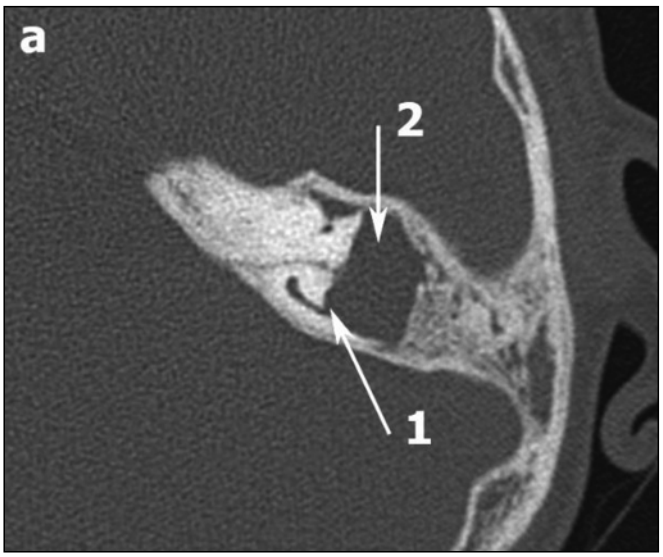

Figure 2. Axial high-resolution CT scan of the temporal bone.
A fistula of the posterior semicircular canal *(arrow 1)* and cholesteatoma in the antrum *(arrow 2)*.

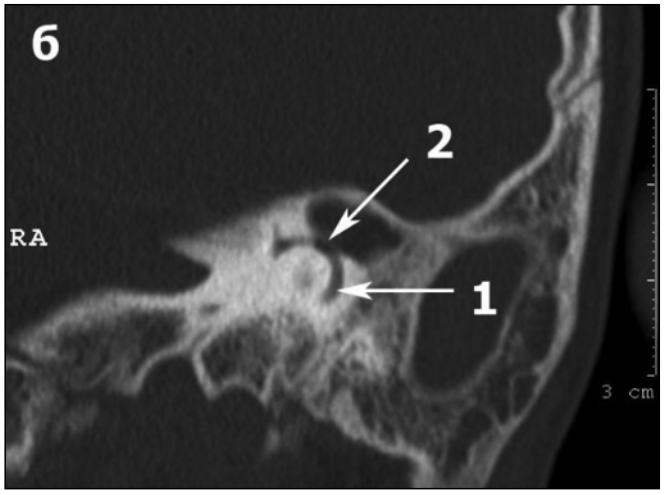

Figure 3. High-resolution CT scan of the temporal bone in the plane of posterior semicircular canal.
The lumen *(arrow 1)* and fistula *(arrow 2)* of posterior semicircular canal.

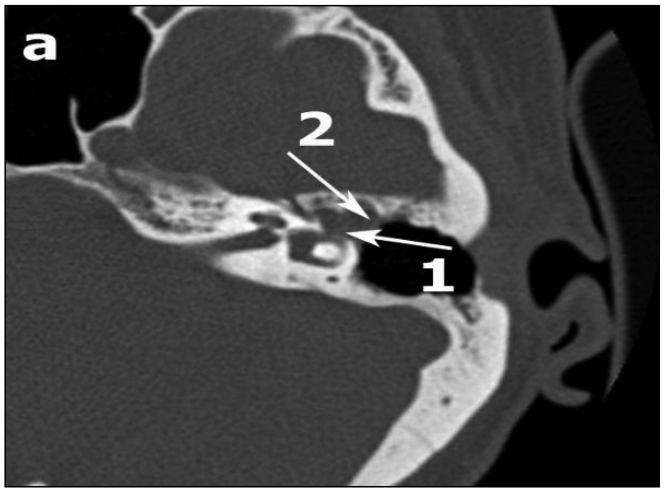

Figure 4. Axial high-resolution CT scan of the temporal bone after canal-wall-down mastoidectomy before the surgical treatment.
A fistula of the lateral semicircular canal *(arrow 1)* and recurrent cholesteatoma *(arrow 2)*.

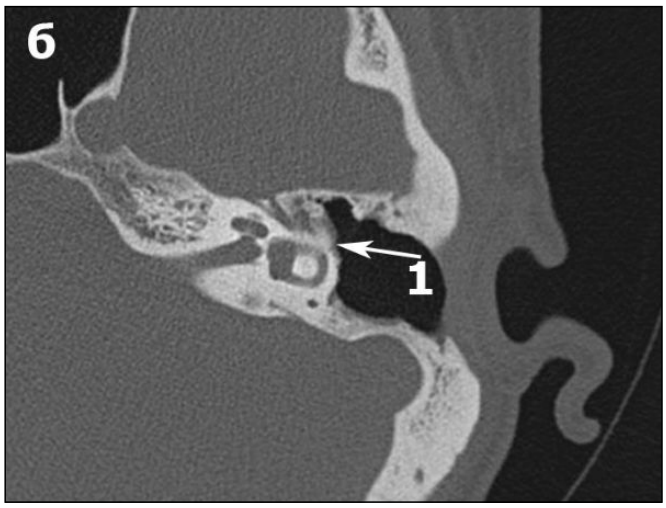

Figure 5. The same patient (fig. 4) CT scan of the temporal bone 12 months later the surgical treatment.
The restoration of integrity of the semicircular canal wall *(arrow 1)*.

Meniere's Disease

PP13

BLOOD HORMONES LEVEL IN PATIENTS WITH MÉNIÈRE'S DISEASE

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Stress has been postulated to trigger or contribute to inner ear pathologies, yet there is little objective evidence.

Materials-Methods: We investigated steroid and pituitary hormones in 42 Ménière's patients between the ages of 29 and 47. The diagnosis was clinically determined on grounds of 2 or more attacks of vertigo lasting at least 20 minutes, followed by intensification of tinnitus and aural fullness, as well as unilateral conductive hearing loss confirmed by audiometry.

All patients were divided into 2 groups. Group 1 embraced 28 people whose attacks of vertigo occurred 2 to 5 times a year. Group 2 included 14 patients during attacks acceleration up to 2-3 times a week. Serum hormones level (cortisol, aldosterone, testosterone, prolactin, growth hormone) was determined by enzyme-linked immunoassay during the attack and in the attack-free interval. The data were compared to those from a control group of 20 healthy volunteers.

Results: The serum cortisol, growth hormone and prolactin were higher in patients of group 1 compared with the control group. The differences were significant ($p < 0.05$) as well as testosterone regression in males (table 1, 2). The patients of group 2 showed significant cortisol decrease during attacks versus the control group ($p < 0.05$) and growth hormone and prolactin increase more expressed than in those with rare attacks. Plasma aldosterone was not elevated during an attack in patients of both groups as compared to plasma aldosterone in the control group. In the attack-free interval the level of the studied hormones showed no difference from the control group data.

Discusion: Thus, the basic differences between the patients of group 1 and those of group 2 during the attack were in the cortisol and prolactin levels. We suppose that low cortisol level in patients of group 2 is rather the result than the cause of frequent attacks of vertigo, but taking into account the glucocorticoids stimulatory effect upon the activity of neurons and the available published indications of cortisol participation in the internal ear homeostasis regulation (Horner K.C., Cazals Y., 2005; Aoki M. et al., 2011), this might contribute to further increasing of the attacks frequency. In our opinion, the significant increase of prolactin in patients of group 2 reflects the change in the mechanisms of its production. It is common knowledge that prolactin synthesis and secretion by adenohypophysis is regulated by hypothalamic inhibitor dopamine (prolactin inhibiting factor) synthesized in the tuberoinfundibular dopaminergic tract. Dopamine reduction in central structures results in blood prolactin rise. The existing experimental, as well as clinical data obtained by way of examining Parkinson disease patients, are indicative of dopamine participation in efferent control of the vestibular signal. It is not inconceivable that hyperprolactinemia revealed in the patients of group 2 reflects dopamine insufficiency, which is a factor reducing the compensation abilities of the vestibular system.

Conclusions: These data provide further evidence for modification of different stress hormones in audiovestibular pathologies, which might provide a valuable prognostic tool in the future.

Keywords: Ménière's disease, vertigo, cortisol, aldosterone, prolactin

Table 1. Serum steroid hormones level in patients with Ménière disease

Hormones	Group 1, during the attack (n=28)	Group 1, in the attack-free interval (n=28)	Group 2, during the attack (n=14)	Group 2, in the attack-free interval (n=14)	Control group (n=20)
cortisol (nmol/l)	728,10 ± 52,21 *, **	416,64 ± 36,95	293,51 ± 38,14 *,**	381,10 ± 32,47	453,68 ±29,39
aldosterone (nmol/l)	0,448 ± 0,022	0,390 ± 0,030	0,456 ± 0,089	0,410 ±0,061	0,424 ±0,026
testosterone (males, nmol/l)	3,68 ± 0,24*,**	9,84 ±0,89	4,46 ± 1,96*,**	10,28 ± 2,15	11,61 ± 1,06
testosterone (females, nmol/l)	0,95 ± 0,23	1,43 ± 0,23	0,94 ±0,28	1,22 ± 0,19	1,38 ± 0,10

Note: An asterisk (*) denotes statistically significant difference versus the control group ($p < 0,05$); two asterisks (**) denote statistically significant difference versus the attack-free interval ($p < 0,05$).

Table 2. Serum pituitary hormones level in patients with Ménière disease.

Hormones	Group 1, during the attack (n=28)	Group 1, in the attack-free interval (n=28)	Group 2, during the attack (n=14)	Group 2, in the attack-free interval (n=14)	Control group (n=20)
Prolactin (males, mME/ml)	196,81 ± 15,30 *	144,92 ±19,09	346,34 ± 56,28 *,**	156 ± 18,98	169,31 ± 15,71
Prolactin (females, mME/ml)	453,52 ± 61,30 *,**	228,05 ±37,05	709,23 ±96,14 *,**	217 ±26,65	280,97 ±27,79
Growth hormone (ng/ml)	1,23 ± 0,31 *,**	0,76 ± 0,12	1,51 ± 0,28 *,**	0,71 ±15	0,61 ± 0,11

Note: An asterisk (*) denotes statistically significant difference versus the control group ($p < 0,05$); two asterisks (**) denote statistically significant difference versus the attack-free interval ($p < 0,05$).

Vestibular Diagnosis-New Tools

PP14

VESTIBULAR DISORDERS IN THE FUNCTIONAL STAGE OF VERTEBRAL ARTERY SYNDROME

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The Purpose: Detection of early diagnostic signs of vertebrobasilar insufficiency (VBI) in patients with cervicocranialgia and functional stage of vertebral artery syndrome.

Materials and methods: Study was carried out using a computer stabilometry. The essence of this method is to register the position and motion of the common center of pressure on a plane bearing upon standing. This method allows to fix minor violations of coordination during exercise testing.

Were examined 60 people. 30 patients with mild to moderate cervicocranialgia without clinical manifestations of VBI and disorder of vestibular function. One of the criteria for selection of patients was the lack of significant changes on testing with Doppler ultrasound of the brachiocephalic arteries. The group included 13 men and 17 women, aged 30 to 55 years. The control group consisted of 30 people of the same age and gender features without clinical manifestations of cervicocranialgia.

All patients were performed stabilographic study on the unit Stabilan-1 using functional tests.

The results were evaluated according to; stabilogram - moving the center of pressure graphics (CD) presented as a function of time for the frontal and sagittal planes. Wherein the time axis is horizontal.

Performance is measured on a scale: poor, fair, good, excellent.

Research data in the main group 5 patients (16.7%) - it is bad, 14 patients (46.7%) - satisfactory, 7 patients (23.3%) - well, 4 patients (13.3%) - excellent.

The same studies in control group showed significant differencies: 12 patients (40%) - well, 18 patients (60%) - excellent.

The Conclusion: The study showed that the instrumental diagnosis of the early stages of cervical pain as part of the pathological changes of the spine without clinical manifestations can detect early signs of disorder of vestibular fuction as a part of spondilogenic vertebrobasilar insufficiency. This method can be used as a part of algorithm of early diagnosis and for prevention of disorders of vestibular fuction.

Keywords: vertebrobasilar insufficiency, stabilometry, vertebral artery syndrome

Treatment

PP15

CHIARY ASSOCIATED PATHOLOGY OF COCHLEOVESTIBULAR SYSTEM

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Vestibular problem in case of modern life becomes very actual. More than 50% of patients with vertigo has peripheral problems, but in this case we can forget about complicated diseases, when central pathology is not on the surface and it is impotent in appointment of treatment.

One of such pathologies is the Chiary malformation. It can increase peripheral problems and complicate diagnostic and treatment.

In our research we get 80 patients with Chiary malformation and peripheral pathology.

The Chiary Malformation (CM) is a congenital development defect and associated with the caudal displacement into foramen magnum of cerebellum and brain stem.

The clinical picture of The Chiari Malformation is polymorphic and includes a combination of cerebellar, spinal, bulbar, cochleovestibular disorders, with symptoms of intracranial hypertension.

The aim of our study is to improve the diagnostic methods for the cochleovestibulary dysfunction, which will lead to more effective treatment and will improve patients' quality of life.

The 42.5% of our patients with CM type I at some point had benign paroxysmal positional vertigo.

Diagnosis of benign paroxysmal positional vertigo with the Chiari malformation is complicated due to the presence of tinnitus and hearing loss, that, as it is known, is not a classic clinic of BPPV.

To our opinion, otolithiasis with CM can occur due to a hypertension-hydrocephalic syndrome, that can possibly

contribute to the violation of the perilymph fluid flow in the vestibular apparatus.

Hence the therapy was based not only on positional maneuverings, but also included central diuretic, venotonic drugs and betahistine hydrochloride (Betaserc).

Earlier, prior to our study 32.5% of our patients were diagnosed with Meniere's disease. Which we could confirm with the help of otoneurologic examination, dehydration tests, videonistagmografy, electrocochleography.

But the presence of CM, identified in the MRI of the brain, does not allow us to talk about "Meniere's disease", as it is most likely secondary.

Traditional therapy of Meniere's disease caused significant improvements and after our correction in accordance with diagnosed Chiari I the patience had the long-term absence of relapses.

25% of patients suffered Sensorineural hearing loss. It was a chronic and progressive in nature, and the existence of a "gruelling" tinnitus, greatly worsened the patients' quality of life.

Sensorineural hearing loss with the Chiari malformation poorly amenable to therapy, and in the case of the combined therapy with venotonic, hormonal and diuretic drugs brought expected significant improvement.

The study confirmed that for the effective treatment of cochleovestibulary dysfunction at various levels of disease the systematic approach and the complex diagnosis is absolutely necessary. Which requires a profound knowledge not only in Otorhinolaryngology but in other medical specialties.

Keywords: Chiary malformation, BPPV, Meniere's disease, hearing loss

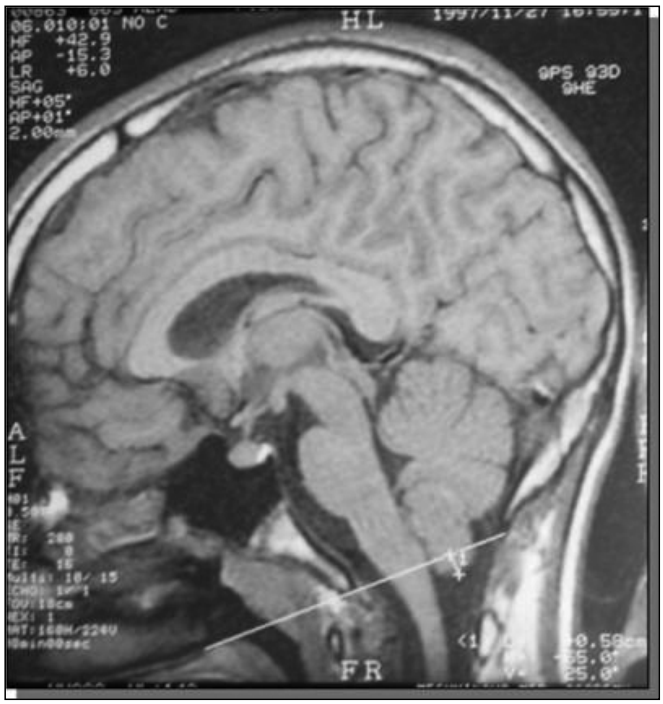

Figure 1. Chiary malformation

BPPV

PP16

MIDDLE AND INNER EAR DISEASES ASSOCIATED WITH BENIGN PAROXYSMAL POSITIONAL VERTIGO

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Objectives: Benign paroxysmal positional vertigo (BPPV) is the most common vestibular disorder in adults. However, the cause of otoconia detachment is unknown in the large majority of cases, which are termed idiopathic BPPV (i-BPPV). Some middle and inner ear diseases may be possible causes of BPPV, since their pathophysiology leads to changes in inner ear structures. We analyzed clinical features of BPPV associated with middle and inner ear disorders.

Methods: We retrospectively reviewed the medical records of 127 patients with BPPV and analyzed 11 cases of them which were associated with middle and inner ear diseases (main group). All patients underwent complete otolaryngological, audiological, and neurotologic evaluation, including nystagmography. Control group included patients with i-BPPV.

Results: The majority of patients (111; 87,4%) had i-BPPV, whereas 11 patients (8,7%) had secondary BPPV, associated with middle and inner ear disorders (se-BPPV) and 5 patients (3,9%) had secondary BPPV, associated with head trauma. Women were affected more frequently than men in a ratio of 1.8:1 in i-BPPV group, but there was no significant sex difference for se-BPPV patients. I-BPPV was most common in patients older than 50 years, and se-BPPV occurred in patients of all ages. Se-BPPV associated with chronic otitis media (3/11), idiopathic sudden sensory hearing loss (ISSHL) (3/11), Ménière's disease (3/11), acute vestibular neuronitis (1/11) and posttraumatic otitis media (1/11). Associated ear diseases in patients with BPPV are presented in Table 1. In both i-BPPV and se-BPPV, the posterior canal was most commonly involved, followed by the lateral canal, and lastly, the anterior canal. Ipsilateral BPPV was observed in all patients with ISSHL and Ménière's disease. The interval between the onset of ISSHL and BPPV was less than 3 days in all patients. The posterior canal BPPV, associated with ISSHL, was effectively cured by one or two repositioning maneuvers. Two of three BPPV cases in patients with chronic otitis media occurred after surgical drilling of the temporal bone. Perhaps traumatic factor played greater role in developing BPPV in the patient with posttraumatic otitis media and in patients, who underwent ear surgery, than influence of inflammation in the middle ear. BPPV, that occurred at ipsilateral side of trauma, involved posterior canal (1 patient with posttraumatic otitis media and 1 patient with chronic otitis media) and BPPV, that occurred at contralateral side of trauma, involved horizontal canal, presenting canalo- and cupulolithiasis (2 patients with chronic otitis media). Treatment durations of BPPV associated with Ménière's disease and chronic otitis media were significantly longer compared to i-BPPV ($p<0.05$, Mann-Whitney U-test).

Conclusions: The mean duration of treatment for BPPV associated with chronic ear diseases (Ménière's disease and chronic otitis media) was longer than for i-BPPV group. A casual relationship exists between middle and inner ear diseases and BPPV.

Keywords: benign paroxysmal positional vertigo, inner ear disease, chronic otitis media

Table 1. Associated ear diseases in patients with benign paroxysmal positional vertigo.

	Number of patients (%)	*Sex (male/ female)*	*Involved semicircular canal (posterior/horizontal/anterior)*
Idiopathic	111 (87,3%)	39/72	97/13/1
Secondary	16 (12,7%)	7/9	9/6/1
- Head trauma	5 (3,9%)	3/2	3/2/0
- Middle ear diseases	4 (3,2%)	2/2	2/2/0
•Chronic otitis media	3 (2,4%)	1/2	1/2/0
•Posttraumatic otitis media	1 (0,8%)	1/0	1/0/0
- Inner ear diseases	7 (5,6%)	2/5	4/2/1
• ISSHL	3 (2,4%)	1/2	1/1/1
• Ménière's disease	3 (2,4%)	1/2	2/1/0
•Vestibular neuronitis	1 (0,8%)	0/1	1/0/0

Imaging

PP17

VASCULAR VERTIGO DOCUMENTED WITH ONLY PERFUSION IMAGING

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Background: Isolated vertigo due to vascular compromise pauses a diagnostic challenge when MRIs including diffusion-weighted images (DWI) do not show acute infarction. The diagnostic value of perfusion-weighted images remains to be explored in acute vascular vertigo.

Method: We analyzed the clinical and radiologic features of 10 patients with acute vascular vertigo that had been confirmed with only perfusion imaging. Follow-up imaging was performed when decided necessary.

Results: All the patients presented acute prolonged (>24 h, n=4) or transient (n=6) vestibular symptoms including vertigo and unsteadiness. Those were isolated in 8, and associated with sensory symptoms in one, and loss of consciousness in another one. All patients except one had vascular risk factors including hypertension (n=6), diabetes mellitus (n=3), atrial fibrillation (n =2), dyslipidemia (n=1), stroke history (n=2) and smoking (n=3). Acute infarction was not found on routine MRIs including DWI in all patients, but, perfusion CT or MRI documented decreased perfusion in the territory of the posterior inferior cerebellar artery (PICA) in 7, PICA and anterior inferior cerebellar artery (AICA) in one, lateral medulla in one, and PICA, brainstem and both posterior cerebral arteries in the remaining one. Angiography documented vascular pathology that corresponded to the areas of perfusion defect in all patients except one. Three of the four patients with prolonged vestibular symptoms showed a neurologic deterioration and follow-up imaging confirmed infarction within two days.

Conclusion: Transient or prolonged vertigo may occur in patients with decreased perfusion in the posterior circulation even without infarction on DWI. Perfusion imaging should be performed in patients with suspected vascular vertigo, especially when routine MRIs including DWI are unrevealing.

Keywords: central vertigo, VBI, brain image, perfusion MRI, vascular vertigo

Vestibular Paroxysmia

PP18

NEUROPHYSIOLOGICAL FINDINGS IN ESTABLISHED VASCULAR COMPRESSION OF THE COCHLEOVESTIBULAR NERVE

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Background: It was recently found that the certain role in the development of the paroxysmal vestibular dysfunction (VD) is played by the factor of the vascular compression of the cochleovestibular nerve (CVN).

Objective: To study neurophysiological findings in the patients with recurrent VD, who have vascular compression of the CVN established by magnetic resonance imaging (MRI 3.0T).

Materials-Methods: 143 patients with vertigo attacks had been examined, including 40 male and 103 female, with mean group age 41.2±8.9 years. All the patients underwent vestibulometry with functional tests. Nystagmus was recorded using electronystagmography («Electronystagmograph»,«Statokyn», Moscow). Brain MRI was performed with isotropic pulse sequence "FIESTA-C" and angiographic sequence (TOF) («DISCOVERY MR750W 3.0T» (GE, USA)).

Results: Brain MRI revealed adherence of vessel to CVN in 43 (30.0%) patients.

All the cases of neurovascular conflict (NVC) are presented by AICA. NVC on the right was found in 20 (46.5%) cases, on the left in 12 (27.9%) cases, bilateral NVC in 11 (25.6%) of 43. In 10 cases combination was observed of NVC with other organic brain pathology. Signs of latent VD were found in 30 (69.7%) of 43 and are presented by the provoked nystagmus in functional tests on the side of the established NVC combined with vestibular hyporeflexia in 10 (33.3%) or vestibular hyperreflexia in 18 (60.0%) on the side of NVC while bithermal calorization. High proportion of position-dependent VD was found in NVC, which comprises 41.3% (18 patients). Such tests as de Klein test in 18 (41.3%) cases and Valsalva maneuver in 13 (30.2%) provoked nystagmus directed to the side of NVC. Nystagmus was detected in more than one functional tests on the side of NVC in 20 cases (46.5%).

Conclusions: Recurrent VD in 30% cases is caused by NVC. NVCs may be depicted in detail by high-field MRI. Detection of latent VD on the side of NVC reflects it's clinical significance and the tendency of the vestibular system to the development of paroxysmal vertigo.

Keywords: vascular compression of the cochleovestibular nerve, neurophysiological findings, vestibulometry, MRI

PP19

VESTIBULAR PAROXYSMIA - NEUROVASCULAR COMPRESSION OF THE VIII NERVE

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Vestibular paroxysmia is a peripheral vestibular disorder characterized by recurrent, brief attacks of rotatory or postural vertigo. The attacks are typically of a short duration and may be associated with change in head position. There may or may not be associated hearing loss and tinnitus. The hearing loss and tinnitus may persist in attack free periods also. Three patients reported with symptoms of tinnitus, giddiness, and spells of brief attacks of vertigo. Magnetic resonance imaging was requested in all three patients. A neurovascular compression of the Vestibulocochlear nerve was reported in all the three cases. One patient responded very well to Carbamazepine administered orally. Since, the audiological or vestibular test findings in patients labeled as having vestibular paroxysmia are not typical or characteristic, Magnetic resonance imaging is a helpful tool in diagnosis of vestibular paroxysmia in the presence of symptoms.

Keywords: Vestibular paroxysmia, postural vertigo, tinnitus, hearing loss, Magnetic resonance imaging

Vestibular Diagnosis-New Tools

PP20

DOES VESTIBULER EVOKED MYOGENIC POTENTIALS (VEMP) HAVE PROGNOSTIC SIGNIFICANCE IN PATIENTS WITH SUDDEN SENSORINEURAL HEARING LOSS WITH OR WITHOUT COMCOMITTANT VERTIGO SYMPTOMS

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Objective: The objective of the study is to investigate whether vestibular evoked myogenic potentials (VEMPs) and distortion product otoacoustic emissions (DPOAEs) have prognostic significance in the follow-up of the patients diagnosed as idiopathic sudden sensorineural hearing loss (ISSNHL) with or without concomittant vertigo symptoms.

Methods: Twenty-seven patients diagnosed as ISSNHL at their first admissions underwent VEMP and DPOAE tests. As a treatment protocol, steroid treatment combined with hyperbaric oxygen therapy was initiated on all patients. At the second month of the treatment, all patients underwent VEMP and DPOAE tests once more. Hearing recovery was evaluated by the improvement in hearing compared to the unaffected contralateral ear.

Results: Mean recovery rate was significantly higher in patients with positive pretreatment VEMP tests, compared to those with negative pretreatment VEMP tests (p=0.023). Mean recovery rate was also found significantly higher in patients with positive posttreatment VEMP tests, compared to those with negative posttreatment VEMP tests (p=0.031). Patients with posttreatment positive DPOAE tests found to have higher mean recovery rate when compared to those with negative DPOAE tests (p<0,001).

Conclusion: We think that in the follow-up of the prognosis of ISSNHL disease, VEMP test may have a prognostic value, while DPOAE test is an important parameter which might be used in the monitorization of the disease.

Keywords: Sudden hearing loss, VEMP, DPOAE, prognostic factors

PP21

OUTCOME EVALUATION OF THE DIZZINESS HANDICAP INVENTORY IN AN OUTPATIENT VESTIBULAR CLINIC

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Background: the DHI is a widely used questionnaire for the evaluation of the self-reported handicap in patients with balance problems.

Objective: to investigate the relationship between the DHI scores and demographic, symptomatic and diagnostic parameters.

Methods: retrospective study in 568 patients with balance problems.

Results: we observed a total of 61.3% of patients with moderate (DHI total score between 30 and 59) to severe (DHI total score between 60 and 100) handicap.

Patients with chronic complaints experience their self-reported disability to a greater extent than acute patients. Moreover, patients suffering from continuous complaints have a larger than patients with shorter symptom duration. The first effect (acute vs. chronic) is primarily caused by emotional factors, the latter effect (symptom duration) is attributable to functional and physical factors, not to emotional aspects. Patients with daily and weekly complaints have larger DHI scores than patients who reported only one episode. Female patients reported larger DHI scores than males. We found no effect of age, diagnosed pathology or reported symptoms on the DHI scores.

Conclusions: the information retrieved from the DHI questionnaire is complementary to the information obtained from clinical investigation and diagnostic tests and therefore is an essential tool in a vestibular clinic.

Keywords: dizziness handicap inventory, vestibular disorders, vestibular symptoms, diagnosis, demographic

PP22

PERIPHERAL VESTIBULAR DYSFUNCTION: NEUROLOGIST'S VIEW

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Background: Vertigo is suffered during the life by 7.8% of population, and annual incidence of vertigo is 5.2% of population (Neuhauser H.K., 2005). There are great number of diseases which cause vertigo. Their diagnosis is hard and requires detailed

analysis of complaints, history, meticulous physical, neurological and otoneurological examination, analysis of instrumental findings. In up to 20% cases cause of paroxysmal vestibular dysfunction (VD) remains unknown.

Objective: To reveal objective signs of peripheral VD in the structure of neurological diseases manifestations using vestibulometry and magnetic resonance imaging (MRI 3.0T).

Materials-Methods: 143 patients with vertigo attacks had been examined, including 40 male and 103 female. All the patients underwent vestibulometry to objectivize VD, brain MRI with isotropic pulse sequence "FIESTA-C" and angiographic sequence (TOF) («DISCOVERY MR750W 3.0T» (GE, USA)).

Results: It was found that in 10 (6.9%) patients with recurrent VD, mean group age 35±7.2 years, MRI revealed signs of the cochleovestibular nerve (CVN) lesion combined with other organic brain changes. So, adherence of blood vessel to CVN combined with demyelination foci was revealed in 4 patients, adherence of blood vessel to CVN combined with ipsilateral CVN scwannoma in 2 patients, CVN demyelination combined with disseminated demyelination foci in the brain matter in 6 cases.

Clinical Case: Female patient V., 52 years. Complaints of uncontrollable facial muscles contractions on the left side, vertigo attacks, unsteadiness while walking. Onset was acute approx. 2 months ago. She was treated for vertebral artery syndrome with no significant effect.

On examination: Facial hemispasm on the left side, left-directed horizontal nystagmus which do not change it's direction while looking to the sides, tendon reflexes high, symmetric, abdominal reflexes absent, moderate coordination impairment..

MRI before contrast injection revealed multiple polymorphic foci of demyelination in the brain hemispheres and the cerebellum hyperintense on T2W and on T2-FLAIR. After OMNISCAN injection some of the foci were enhanced, facial nerves were not enhanced. MRI examination with isotropic pulse sequence "FIESTA-C" revealed blood vessel adherence to the facial and CVN trunks on the left side. According to vestibulometry Findings: horizontal nystagmus to the left side, while de Klein test it's frequency and amplitude increase, while caloric stimulation – vestibular hyporeflexia on the left side, impaired vestibuloocular reflex suppression.

Conclusions: Combination of CVN vascular compression with other degenerative or inflammatory brain changes was noticed in 6.9% cases. To clarify leading role of CVN vascular compression in the disease presentation, it is necessary to carry out vestibulometric study with functional tests.

Keywords: vestibulometry, magnetic resonance imaging, demyelination, cochleovestibular nerve, vascular compression

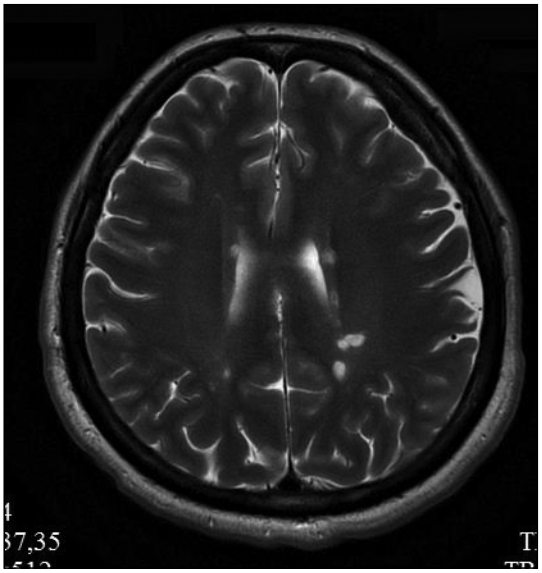

Figure 1. Female patient V. Foci of demyelination in the brain matter.

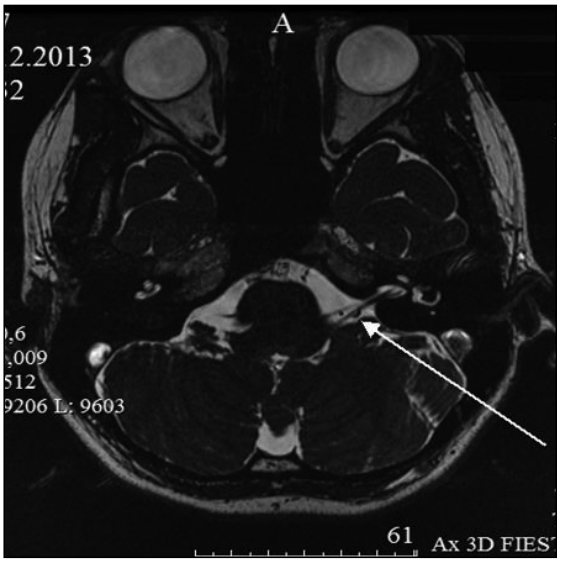

Figure 2. Adherence of the anterior inferior cerebellar artery to the facial and cochleovestibular nerves trunks on the left side.

Acute Vestibular Disorders

PP23

LEVELS OF ANXIETY AND DEPRESSION IN PATIENTS WITH VERTIGO AS A PART OF THE VERTEBRAL ARTERY SYNDROME

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Purpose of the study: to determine the level of anxiety and depressive disorders in people with early signs of vertebral artery syndrome. Reveal hidden affective disorders in the early stages of the disease.

Materials and methods: The main and most common method of diagnosis of anxiety level are physiological tests, questionnaires, such as the Hospital Anxiety and Depression Scale,

the Spielberg Anxiety test, Beck questionnaire. The diagnosis of vertebral artery syndrome and stage of the process was confirmed by clinical neurological examination and Doppler ultrasound test of the brachiocephalic vessels.

Were examined 56 patients aged 23 to 39 years with early signs of vertebral artery syndrome. At the time of passing the test patients were admitted in the hospital from one to nine days. Patients were divided into two subgroups of 28 people, matched by sex and age.

Results: First subgroup included patients with complains of dizziness when changing body position, pain in the neck. Doppler ultrasound of the brachiocephalic vessels found mild decrease in the rate of blood flow in the vertebral and basilar arteries in 17.8% of patients (5 persons) in this subgroup. The results of psychophysical tests of the second subgroup shown in Table 1.

In the second subgroup, along with complaints of dizziness, headaches and neck pain, patients are referred to the lowering of mood, inner tension, inability to relax. Ultrasound scanning of brachiocephalic vessels demonstrate decrease in the rate of blood flow in the vertebral and basilar arteries mild to moderate in 53.6% of patients. The results of psychophysical tests of the second subgroup shown in Table 2.

Conclusion: In the initial stages of development of vertebral artery syndrome, despite the lack of complaints about the reduction of the background mood, patients characterized by hidden anxiety and minor depressive symptoms. As the disease progresses and hemodynamic changes increases, they demonstrate an increased levels of anxiety and depressive disorders to a greater extent to moderate, indicating the need for more detailed psychological examination and preventive correction of affective disorders in patients with functional stage of vertebral artery syndrome.

Keywords: anxiety, depression, vertebral artery syndrome

Table 1. The results of psychophysical tests of the first subgroup

level of changes	*the Hospital Anxiety Scale*	*the Hospital Depression Scale*	*Spielberg Situative Anxiety test*	*Spielberg Individual Anxiety test*	*Beck questionnaire*
light	78,6 %	100%	75%	67,8%	71,4%
mild	21,4%	-	25%	32,2%	28,6%

Table 2. The results of psychophysical tests of the second subgroup

levels of changes	*the Hospital Anxiety Scale*	*the Hospital Depression Scale*	*Spielberg Situative Anxiety tes*	*Spielberg Individual Anxiety tes*	*Beck questionnaire*
Light	21,4 %	71,4%	25%	-	25%
mild	64,2%	28,6%	60,7%	71,4%	75%
severe	14,4%	-	14,3%	28,6%	-

Vestibular Diagnosis-New Tools

PP24

INFARCTION OF THE LABYRINTH - DIAGNOSIS AND MANAGEMENT

Mario Milkov[1], Aleksandar Karapanchev[2], Plamen Nedev[1], Aleksandar Rainov[2], Tzvetan Tonchev[1], Ivan Tzenev[2]
[1]Medical Univerity " Prof.. P. Stoianov" Varna, FDM
[2]Medical University of Sofia

Recently, inner ear disorders represent a common pathology faced by neurotologists worldwide. The infarction of the labyrinth is an acute disorder of labyrinthine circulation due to insufficient blood, oxygen and glucose supply. This is manifested by severe otoneurologic symptoms and requires urgent precise diagnosis and timely management. Isolated labyrinthine infarction could be a harbinger of anterior inferior cerebellar artery ischemic stroke. Labyrinthine infarction could be diagnosed by x-ray and neurotologic examinations such as pure tone audiogram thresholds, spontaneous nystagmus, summating potentials/action potentials on electrocochleography, interaural amplitude difference on the vestibular-evoked myogenic potential test, canal paresis and fixation index on the bithermal caloric test, and gain on oculomotor tests. During the period from 2010 to 2014, 27 patients with acute labyrinthine infarction were hospitalized in the Department of Otorhinolaryngology, Tsaritsa Giovanna University Hospital of Sofia, Medical University of Sofia. There were 20 females and 7 males aged between 41 and 58 years at a mean age of 47±7,1 years. These patients underwent a series of otoneurological tests and urgent conservative treatment. Our results revealed the relative importance of the single diagnostic methods as well as the gradually enhanced effectiveness of the individualized antithrombotic and neurone-protective therapy. Vestibular rehabilitation started early and also contributed to patient's improvement.

Keywords: inner ear, electrocochleography, vestibular rehabilitation

How I Do It Sessions

PP25

VESTIBULAR REHABILITATION OF PATIENTS AFTER STAPEDECTOMY BY NONINVASIVE NEUROMODULATION

Fedor Syroezhkin[1], Alexandra Guseva[2], Vladimir Dvorianchikov[1]
[1]Military Medical Academy
[2]Pirogov Russian National Research Medical University

In spite of delicate techniques in modern approaches to stapedectomy there are vestibular disturbances in some patients with otosclerosis after surgery. Vertigo or vegetative symptoms are rare but imbalance or gait problems can exist for several days or weeks. In those cases patients could be considered as a model of unilateral peripheral disturbances in vestibular system due to sensory asymmetry from labyrinths. In the absence of a fully functional vestibular system, the brain is unable to correctly integrate inherently ambiguous visual and proprioceptive cues. Patients can

experience multiple problems with posture control and movement, including unsteady balance, abnormal gait, and various balance-related difficulties.

An electrotactile vestibular substitution system (ETVSS; BrainPort balance device) transmits head position through tactile sensation of the tongue. As is shown, the use of this device is an effective intervention for individuals with bilateral peripheral vestibular loss with statistically significant improvements in balance, posture, and gait.

The aim of this study was to investigate vestibular symptoms and their effect on the balance in otosclerosis patients undergoing stapedotomy operations with and without vestibular rehabilitation by the ETVSS.

Fifty-three patients (sixty-seven ears) undergoing stapedotomy were included in the study. Randominosly thirty-seven patients had vestibular rehabilitation and other (thirty patients) had no any intervention.

The BrainPort balance device consists of a controller, which hangs around the user's neck; an intraoral device, which adheres to the user's tongue. An accelerometer detects acceleration caused by either gravity or movement. The controller uses this information to derive head position and generate a corresponding stimulus pattern, which is sent to electrodes on the tongue. Subjects were instructed verbally to place the electrode on the top and front part of the tongue. Subjects trained with eyes closed and attempted to maintain static balance by keeping the stimulus centered on the tongue. The subject then practiced maintaining that posture for 20 minutes twice a day. Subjects progressed to the next level when they were able to perform a trial with their eyes closed without assistance to maintain balance. Total period of rehabilitation was 10 days in both groups.

A protocol of computerized posturography was used to analyse the static balance in patients preoperatively, and in the first postoperative two weeks. The dynamic balance was examined by dynamic gait test as a functional outcome measure that was used to assess the patient's ability to maintain gait during eight different tasks.

Preoperatively, all patients were asymptomatic when considering the vestibular system; however, eight of them got low posturography scores on vestibular examination. Postoperatively 86% of the patients had vestibular complaints in variable severity. All patients from rehabilitation group recovered by the third day. The patients without intervention recovered by the end of postoperative second week. A significant drop in static balance and dynamic gait scores was encountered at the first week testing (Student's T-test, P = 0.001).

Thus, electrotactile vestibular substitution system can be considered an effective method of vestibular rehabilitation in patients after stapedotomy causes with gait and balance disturbances.

Keywords: vestibular rehabilitation, otosclerosis, stapedectomy, dynamic gait index, computerised posturography, BrainPort balance device

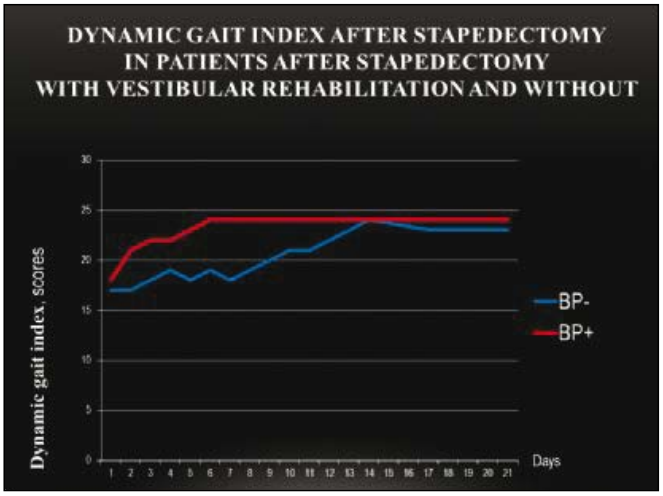

Figure 1. Dynamic gait index evolution

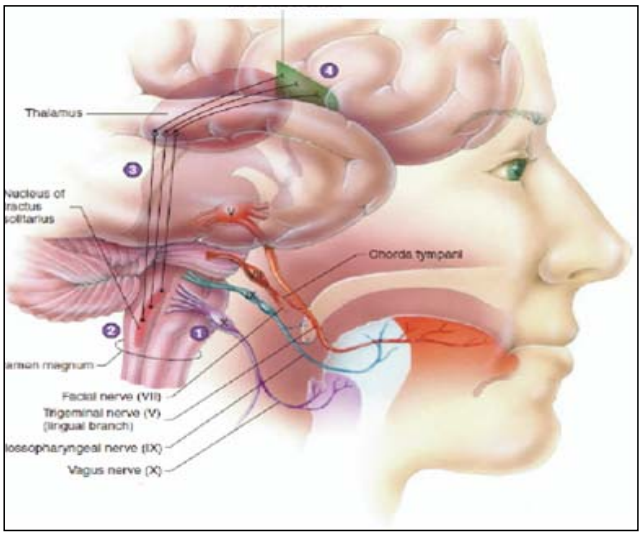

Figure 2. neuroanatomy issue

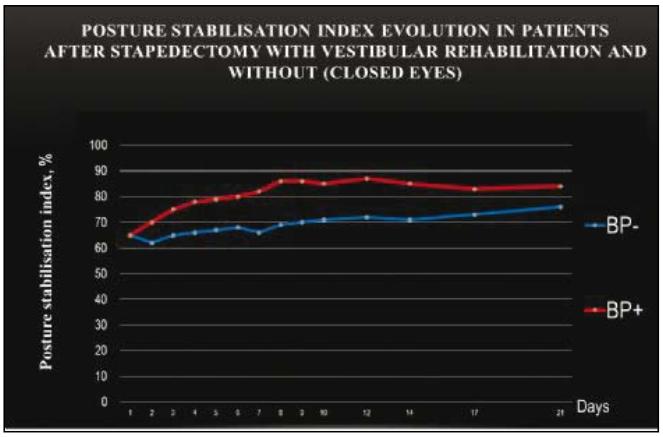

Figure 3. Posture stabilisation index evolution

Figure 4. Rehabilitation procedure

Figure 5. Rehabilitation procedure 2

BPPV

PP26

COMPARISON OF EFFICACY OF THREE POPULAR REPOSITIONING MANEUVERS IN THE TREATMENT OF BENIGN PAROXYSMAL POSITIONAL VERTIGO OF POSTERIOR SEMICIRCULAR CANAL

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[i]Introduction: BPPV is the most common cause of peripheral vertigo characterized by sudden onset of rotational vertigo in certain positions of head. The most commonly affected canal is the posterior semicircular canal. The diagnosis is based on typical history and clinical findings of rotational short-lasting nystagmus on Dix-Hallpike test. Management is in the form of special particle repositioning maneuvers like Epley's maneuver, Semont's maneuver and Brandt-Daroff exercises.

Objectives:

1. To analyze the demographic profile and etiology in patients of BPPV

2. To evaluate and compare efficacy of the three maneuvers in the treatment of BPPV.

Study Design: Prospective Cohort Study.

[b]Setting: Tertiary care rural hospital of teaching institute in Pune, India.

Material and Methods: Patients coming with positional vertigo to OPD were subjected to thorough history and examination as per preformed Performa. Patients with positive Dix-Hallpike test were included. The degree of handicap is assessed by Dizziness Handicap Inventory. The nystagmus was evaluated and recorded on the VNG. 30 patients having confirmed BPPV of posterior semi-circular canal were randomly treated with one of the three maneuvers i.e. Epley's, Semont's and Brandt-Daroff exercises (10 subjects to each treatment mode). Patients followed up on 7th, 14th and 30th day and were re-assessed with a questionnaire and Dix-Hallpike test. The outcomes were classified as Type I, II & III as follows.

TYPE I no symptoms no nystagmus

TYPE II no symptoms nystagmus present

TYPE III symptoms present nystagmus present

Observations: Out of 30 patients presenting with BPPV 53 % (16) were males and 47 % (14) females. 10 %(3) were in age group of 16-30 years, 70 % (21) in 31-60 years and 20 % (6) in age group >60 years. 56% (17) were sedentary, 34 % (10) housewives and 10 % (3) heavy workers. 16% (4) of patients had history of BPPV in past. 19% (6) had history of trauma,12% (4) of vestibular disease, 3% (1) of ear/dental surgery and 50% (15) were idiopathic. On examination 22 patients (73%) had right sided and 8(26%) had left sided posterior semicircular canal BPPV. Percentage of patients treated with Epley's maneuver showing Dix-Hallpike Test Type I were 84.84% and type II were 3%, those treated with Semont's maneuver showing Dix-Hallpike Test Type I were 93.93% and type III were 3% and those patients treated with Brandt-Daroff exercises showing Dix-Hallpike Test Type I were

83.33% and type III were 16.67%.The distribution of patients showing symptoms like instability (post-BPPV syndrome) in the three groups was 21.18% in Epley's maneuver, 24.47% in Semont's maneuver and 26.67% in Brandt-Daroff exercises.

Conclusion: The majority of the cases are males and sedentary. Also, the most commonly affected age group is 31-60 years. Right ear is more commonly affected, the most common cause being idiopathic. The two maneuvers- Epley's and Semont's are of equal efficacy in management of BPPV. Brandt-Daroff exercises have a lower efficacy than the other two maneuvers.

Keywords: B.P.P.V, Epley, Semont, Brandt-Daroff, Maneuver

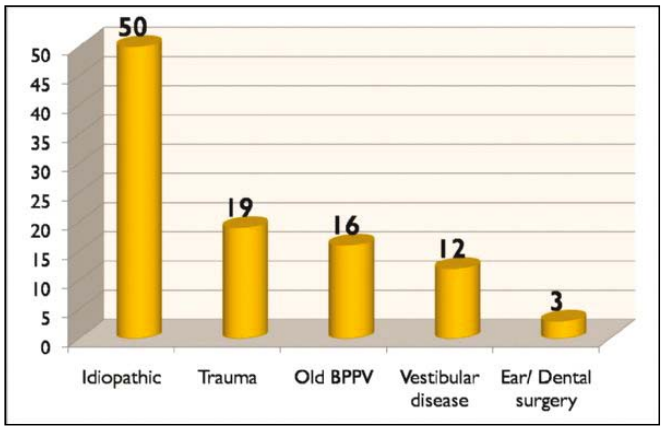

Figure 1. Etiology wise distribution in percentage of 30 patients of B.P.P.V.

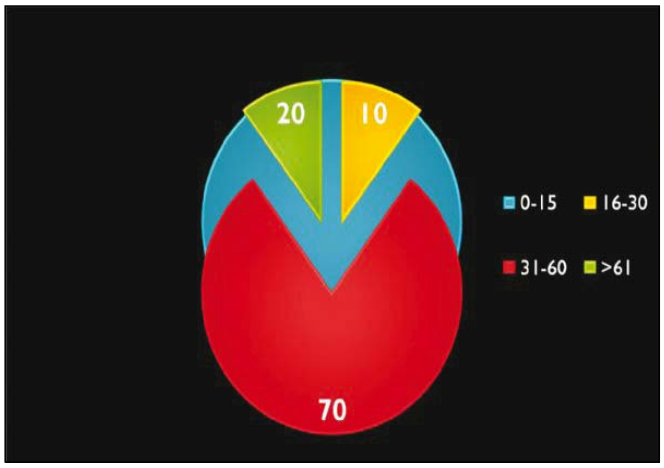

Figure 2. Percentage of Age distribution in 30 patients of posterior canal B.P.P.V

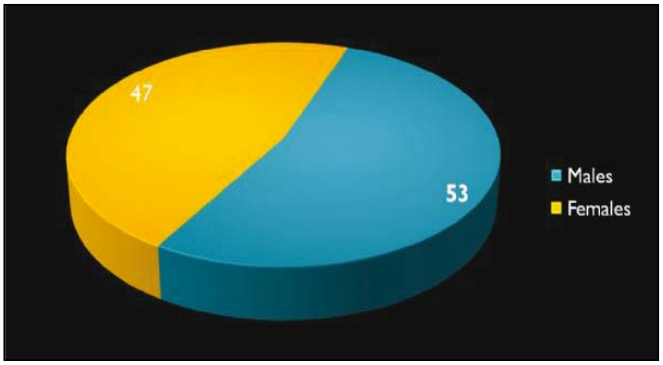

Figure 3. Percentage of sex distribution in 30 patients of posterior canal B.P.P.V.

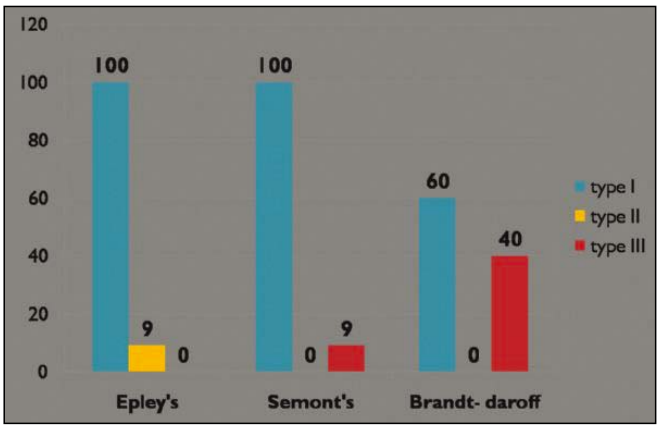

Figure 4. Results in percentage, of the three treatment maneuvers on the 14th day

Table 1. Results of Dix-Hallpike Test (post treatment) in the three treatment maneuvers

Type	*Epley's maneuver*	*Semont's maneuver*	*Brandt-Daroff exercises*
Type I	84.84 %	93.93%	83.33%
Type II	3%	0	0
Type III	0	3%	16.67%

Few patients didnot follow-up viz;Epley's maneuver=3 Semont' maneuver=2 Brandt-Daroff exercise=0

Table 1. Classification(type) of results of Dix-Hallpike test

Type I	No symptoms	No nystagmus
Type II	No symptoms	Nystagmus present
Type III	Symptoms present	Nystagmus present

PP27

WHAT BENEFITS DOES DIX- HALPIKE MANEUVER YIELD?

Süheyl Haytoğlu, Abdurrahman Bozkurttan, Soner Kadıköylü, Gökhan Kuran, Osman Kürsat Arıkan

Adana Numune Training and Research Hospital, Ear, Nose and Throat Clinic, Adana, Turkey

Benign paroxysmal positional vertigo(BPPV) is a disease accampanied by presents with typical symptoms and signs that can be diagnosed easily with the Dix-Hallpike maneuver. The aim of our study is to draw attention for diagnosing and treating this situation rapidly by canalizing the patient suffering from vertigo and BPPV to an ear-nose-throat (ENT) specialist by the clinics dealing with vertigo for an appropriate management. In this way, the unnecessary health spendings of our country and increasingly around the world for diagnosis and treating BPPV can be prevented and the further investigations carried out unnecessarily on these patients admitted to hospitals will be decreased.Also health will be restored in a short period. 38 patients with a diagnosis of Posterior BPPV treated with Dix-Hallpike maneuver are included in our study. A detailed history physical examination were conducted on the patients. The patients underwent Epley maneuver for treatment immediately after the diagnosis and implementation of instructions to be followed by the patient during the week was given. After epley maneuver was performed,at end of the first week and the end of the first month, the complaints of patients were investigated. 4 patients whose complaints were ongoing in the end of the first week, underwent Dix-Hallpike maneuver and Epley maneuver again. At the end of the first month, the patients were successfully treated and they stated that they felt more confident. The lab tests and imagings done by other clinic that the patient applied before the ENT clinics were questioned with their costs to show the unnecessary spendings.

Keywords: Benign paroxysmal positional vertigo, Dix-Hallpike maneuver, Epley maneuver

Table 1. The lab tests and imagings of the patients with their costs.

Tests	*Number*	*Cost per patient(TL)*	*Total cost(TL)*
Complete blood test	22	3	66
Blood Biochemistry	22	15	330
Brain Computerised tomography	26	55	1430
Brain MRI	10	65	650
Doppler USG	4	21	84
ECG	4	3	12
Pure tone Odiometry	4	3	12
Chest X-ray	2	6	12
Total			2600

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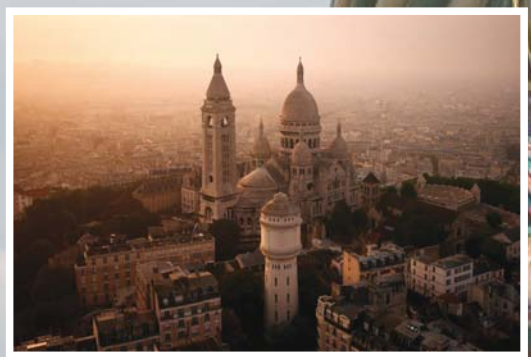

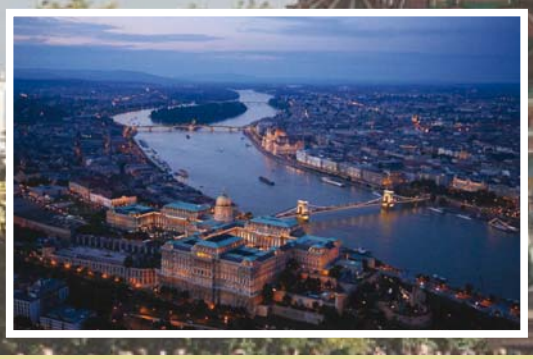

Time	: GMT +3 (Last Sunday in October - last Sunday in March: GMT +4)
Capital	: Moscow
Location	: 55°45′ N 37°37′ E
Altitude	: 150-200 m
Main river	: Moskva (503 km), a tributary of the River Oka
Area	: 1,081.00 km^2
Population	: 10,409,200 (within the city area, 2005)
Population density	: 8,537.2 people/km^2
Postal code	: 119992-123182
Dialing code	: +7 495, +7 499 (digital network)

WEATHER (AVERAGES)

Month	High °C (°F)	Low °C (°F)	Precipitation cm
January	-6.1 (21)	-11.7 (11)	3.56 (1.40)
February	-4.4 (24)	-11.1 (12)	2.79 (1.10)
March	1.1 (34)	- 5.6 (22)	3.30 (1.30)
April	9.4 (49)	1.1 (34)	3.81 (1.50)
May	17.2 (63)	6.7 (44)	5.08 (2.00)
June	20.6 (69)	10.6 (51)	6.60 (2.60)
July	21.7 (71)	12.8 (55)	8.13 (3.20)
August	20.0 (68)	11.1 (52)	7.11 (2.80)
September	13.9 (57)	6.1 (43)	5.84 (2.30)
October	7.2 (45)	0.6 (33)	5.08 (2.00)
November	0.0 (32)	-4.4 (24)	4.32 (1.70)
December	-3.9 (25)	-8.9 (16)	4.32 (1.70)

Currency : Russian Ruble (currently $1 is very approximately 30ru.)
Official Languages : Russian. 35 other languages co-official in various regions
Government : Federal Semi-Presidential Constitutional Republic

President	*Vladimir Putin*
Prime Minister	*Dmitry Medvedev*
Chairman of the Federation Council	*Valentina Matviyenko*
Chairman of the State	*Sergey Naryshkin*

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PUBLIC HOLIDAYS

Holiday	Date
New Year's Day	January 1,2
Christmas Day	January 7
Defenders of the Fatherland Day	February 23
International Women's Day	March 8
Labor Day/May Day	May 1,2
Victory Day	May 9
Independence Day	June 12
Day of Accord and Reconciliation (formerly Day of the Great October Revolution)	November 7
Constitution Day	12 December

Patron Saint : Saint George
City founded : 1147 by Yuri Dolgoruky, Grand Duke of Kiev
City anniversary : 4 September
Historic status : Principality (c1300 - c1480)
Capital of Russia (c1480 - 1703)
Capital of the USSR (1922 - 1991)
Capital of the Russian Federation (1991 onwards)

MOBİLES

Most European GSM mobile phones will work in Moscow, check with your network provider at home to see if you are covered. Calls will be expensive, however, and if you are staying in the city for any length of time you may want to buy a Russian SIM-card for your phone.
If you don't hold a Russian passport, you will probably only be able to get a network service that requires you to buy top-up cards, available from the many mobile phone shops all around the city, as well as supermarkets, kiosks etc. The main networks are Megafon, Beeline and MTS, all of whom offer good coverage (in Moscow you can even use your mobile on the metro), and similar prices. Tele2 is the cheapest network, but its coverage can be a little patchy. A SIM-card should cost around $10, and should have some talk time included

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ВЕРТИГОЛИТИК ВЫСОКОЙ ПРОБЫ
24 КАРАТА ЧИСТОЙ **ОРИГИНАЛЬНОСТИ**

Бетасерк® 8 мг, 16 мг. МНН: *бетагистин.* ***Фармакологические свойства:*** *частичный агонист Н1-гистаминовых и антагонист Н3-гистаминовых рецепторов вестибулярных ядер ЦНС.* ***Показания к применению:*** *синдром Меньера, характеризующийся следующими основными симптомами: головокружение (сопровождается тошнотой/рвотой); снижение слуха (тугоухость); шум в ушах. Симптоматическое лечение вестибулярного головокружения (вертиго).* ***Противопоказания:*** *феохромоцитома; повышенная чувствительность к любому из компонентов препарата. Бетасерк® не рекомендуется для применения у детей в возрасте до 18 лет в связи с недостаточностью данных по эффективности и безопасности.* ***С осторожностью:*** *пациенты с бронхиальной астмой, язвенной болезнью желудка и/или двенадцатиперстной кишки требуют тщательного наблюдения в период лечения.* ***Применение при беременности и в период грудного вскармливания:*** *Беременность. Имеющихся данных о применении бетагистина беременными женщинами недостаточно. Потенциальный риск для человека неизвестен. Бетагистин не должен использоваться во время беременности за исключением случаев абсолютной необходимости. Лактация. Неизвестно выделяется ли бетагистин с грудным молоком. Не следует принимать препарат во время грудного вскармливания. Вопрос о назначении лекарственного препарата матери должен решаться только после сопоставления пользы грудного вскармливания с потенциальным риском для грудного ребенка.* ***Способ применения и дозы:*** *внутрь, во время еды. Доза препарата для взрослых составляет 24-48 мг бетагистина в день. Бетасерк® 8 мг следует принимать по 1-2 таблетки 3 раза в день. Бетасерк® 16 мг следует принимать по ½ -1 таблетке 3 раза в день. Таблетку можно разделить на две равные части (см. инструкцию по медицинскому применению). Дозу следует подбирать индивидуально в зависимости от реакции на лечение. Улучшение иногда наблюдается только через несколько недель лечения. Наилучшие результаты иногда достигаются после нескольких месяцев лечения. Имеются данные о том, что назначение лечения в начале заболевания предотвращает его прогрессирование и/или потерю слуха на более поздних стадиях.* ***Побочное действие:*** *тошнота; диспепсия; головная боль.* ***Перечень всех побочных эффектов представлен в инструкции по медицинскому применению. Передозировка:*** *Известно несколько случаев передозировки препарата. Рекомендуется симптоматическое лечение.* ***Полную информацию о передозировке см. в инструкции по медицинскому применению. Взаимодействие с другими лекарственными средствами:*** *Данные in vitro показали ингибирование метаболизма бетагистина под действием препаратов, которые ингибируют моноаминоксидазу (МАО), включая МАО подтипа В (например, селегилин). Следует соблюдать осторожность при одновременном назначении бетагистина и ингибиторов МАО (включая МАО-В).* ***Влияние на способность к управлению автомобилем и другими механизмами:*** *Бетагистин не влияет или незначительно влияет на способность управлять транспортными средствами и работать с механизмами, в клинических исследованиях нежелательные реакции, которые могли бы повлиять на такую способность, не выявлены.* ***Условия отпуска из аптек:*** *по рецепту.* ***Полная информация по препарату представлена в инструкции по медицинскому применению.*** *ИМП от 27.08.2014*

Бетасерк® 24 мг. МНН: *бетагистин.* ***Фармакологические свойства:*** *частичный агонист Н1-гистаминовых и антагонист Н3-гистаминовых рецепторов вестибулярных ядер ЦНС.* ***Показания к применению:*** *синдром Меньера, характеризующийся следующими основными симптомами: головокружение (сопровождается тошнотой/рвотой); снижение слуха (тугоухость); шум в ушах. Симптоматическое лечение вестибулярного головокружения (вертиго).* ***Противопоказания:*** *феохромоцитома; повышенная чувствительность к любому из компонентов препарата. Бетасерк® не рекомендуется для применения у детей в возрасте до 18 лет в связи с недостаточностью данных по эффективности и безопасности.* ***Применение при беременности и в период грудного вскармливания:*** *Беременность. Имеющихся данных о применении бетагистина беременными женщинами недостаточно. Потенциальный риск для человека неизвестен. Применение бетагистина во время беременности допускается, если польза от применения для матери превышает потенциальный риск для плода. Лактация. Неизвестно выделяется ли бетагистин с грудным молоком. Не следует принимать препарат во время грудного вскармливания. Вопрос о назначении лекарственного препарата матери должен решаться только после сопоставления пользы грудного вскармливания с потенциальным риском для грудного ребенка.* ***С осторожностью:*** *пациенты с бронхиальной астмой, язвенной болезнью желудка и/или двенадцатиперстной кишки требуют тщательного наблюдения в период лечения.* ***Способ применения и дозы:*** *внутрь, во время еды. Доза препарата для взрослых составляет 48 мг бетагистина в день. Бетасерк® 24 мг следует принимать по 1 таблетке 2 раза в день. Таблетку можно разделить на две равные части (см. инструкцию по медицинскому применению). Дозу следует подбирать индивидуально в зависимости от реакции на лечение. Улучшение иногда наблюдается только через несколько недель лечения. Наилучшие результаты иногда достигаются после нескольких месяцев лечения. Имеются данные о том, что назначение лечения в начале заболевания предотвращает его прогрессирование и/или потерю слуха на более поздних стадиях.* ***Побочное действие:*** *тошнота; диспепсия; головная боль.* ***Полный перечень побочных эффектов представлен в инструкции по медицинскому применению. Передозировка:*** *Известно несколько случаев передозировки препарата. Рекомендуется симптоматическое лечение.* ***Полную информацию о передозировке см. в инструкции по медицинскому применению. Взаимодействие с другими лекарственными средствами:*** *Данные in vitro показали ингибирование метаболизма бетагистина под действием препаратов, которые ингибируют моноаминоксидазу (МАО), включая МАО подтипа В (например, селегилин). Следует соблюдать осторожность при одновременном назначении бетагистина и ингибиторов МАО (включая МАО-В).* ***Влияние на способность к управлению автомобилем и другими механизмами:*** *Бетагистин не влияет или незначительно влияет на способность управлять транспортными средствами и работать с механизмами, в клинических исследованиях нежелательные реакции, которые могли бы повлиять на такую способность, не выявлены.* ***Условия отпуска из аптек:*** *по рецепту.* ***Полная информация по препарату представлена в инструкции по медицинскому применению.*** *ИМП от 27.08.2014*

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